UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF IOWA
CENTRAL DIVISION

RENE JUNK, as Parent and Next Best Friend of T.J., a Minor, Plaintiff, vs. TERMINIX INTERNATIONAL COMPANY LIMITED PARTNERSHIP, THE DOW CHEMICAL COMPANY, DOW AGROSCIENCES LLC, HAROLD OBRECHT, an individual, JIM BRENEMAN, an individual, and SURECO, INC., Defendants.	)	Case No. 4:05-cv-00608

DEFENDANTS DOW AGROSCIENCES LLC AND THE DOW CHEMICAL COMPANY'S MEMORANDUM IN SUPPORT OF MOTION TO EXCLUDE THE EXPERT CAUSATION TESTIMONY OF CYNTHIA BEARER, M.D. AND MOHAMED ABOU-DONIA, PH.D. AND MOTION FOR SUMMARY JUDGMENT

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Defendants Dow AgroSciences LLC and The Dow Chemical Company (collectively "DAS") move the Court pursuant to Fed. R. Evid. 104(a) to exclude Plaintiff's causation expert witnesses Cynthia Bearer, M.D. and Mohamed Abou-Donia, Ph.D. Because Plaintiff is unable to offer any reliable proof of medical causation under Fed. R. Evid. 702 without this expert testimony, Plaintiff cannot satisfy the essential element of causation and, therefore, DAS is entitled to summary judgment as a matter of law.

INTRODUCTION

Plaintiff alleges that exposure to the insecticide Dursban* L.O. during a series of routine crack and crevice applications by Terminix International Company Limited Partnership ("Terminix") caused various adverse medical conditions in her[1] son, T.J., including cerebral palsy and neurodevelopmental delay. Plaintiff focuses her causal claims on the effects of Dursban's active ingredient, chlorpyrifos. To succeed in her claims, Plaintiff must demonstrate a causal link between the alleged exposure to chlorpyrifos and T.J.'s cerebral palsy and resulting neurodevelopmental delay. Plaintiff offers the testimony of Cynthia Bearer, M.D., a medical doctor, and Mohamed Abou-Donia, Ph.D., an agricultural chemist and toxicologist, in an effort to meet that burden.

Plaintiff's experts' methods and testimony are unreliable, irrelevant and will not assist the trier of fact. Plaintiff's experts' methods fail to establish that chlorpyrifos is even *capable* of causing cerebral palsy or neurodevelopmental delay, let alone prove that T.J.'s alleged exposure was *in fact* the cause of his unfortunate medical conditions. Drs. Bearer and Abou-Donia use

* Dursban is a registered trademark of Dow AgroSciences LLC.

[1] For convenience, DAS uses the feminine personal pronoun and possessive forms to denote "Rene Junk, as Parent and Next Best Friend of T.J., a Minor," suing in her representative capacity as such, without suggesting that Rene Junk has any personal claim or is a plaintiff in her personal or individual capacity.

unscientific methodologies and rely on inapplicable data to reach their speculative conclusions about chlorpyrifos and its potential effects. Plaintiff's experts have relied on contradictory studies and inappropriately extrapolated data from animal studies that have no correlation to human predictability for cerebral palsy or disease in general. Drs. Bearer and Abou-Donia have also failed to exclude other possible causes of T.J.'s cerebral palsy and failed to consider the dose or exposure level allegedly received by Rene Junk and/or T.J.

This Court should exercise its gatekeeping function and exclude the testimony of Drs. Bearer and Abou-Donia on the issue of causation. Plaintiff's failure to submit competent and reliable expert testimony on the issue of medical causation is fatal to her claims, and therefore DAS' motion for summary judgment should be granted.

STATEMENT OF FACTS

A. Plaintiff's claims.

Plaintiff in this personal injury case alleges that Defendant Terminix periodically treated the Junks' home beginning in March 1992 and continuing until late 1994 with DAS' Dursban L.O. insecticide, in a crack and crevice application; that T.J. was exposed *in utero* to Dursban L.O. and during infancy; and that these exposures caused T.J.'s cerebral palsy and resulting developmental delay.[2]

B. Factual background.

Rene Junk became pregnant with her first child in 1992, but in her sixth month of pregnancy doctors discovered a large tumor on her umbilical cord called a chorioangioma. Expert Report of Dr. John Graham ("Graham Rep.") at ¶ 49, Ex. B to Declaration of Dr.

[2] At all times pertinent hereto, Dursban L.O., and its active insecticidal molecule, chlorpyrifos, were registered for such residential uses with the EPA pursuant to the comprehensive federal statute regulating all pesticides sold and used in the United States, the Federal Insecticide, Fungicide and Rodenticide Act ("FIFRA"), 7 U.S.C. §§ 136-136y, and its implementing regulations. *See* Declaration of Kenneth D. Racke [Document 132-3 at 26-29.]

Graham, The Dow Defendants' Evidentiary Appendix in Support of Motion to Exclude Expert Causation Testimony ("App."), Ex. 1. Her doctors were concerned and did not know how her child would be affected by this large, rare tumor. *Id.*

Rene Junk delivered T.J. on August 28, 1992, approximately two (2) months prematurely. Graham Rep. at ¶ 1, 50. T.J. immediately required resuscitation, and his initial blood gas tests revealed that he had been deprived of oxygen *in utero* and suffered from brain asphyxia. *Id.*; *see also* Expert Report of Dr. Anthony Scialli ("Scialli Rep.") at 3, 9, Ex. B to Declaration of Dr. Scialli ("Scialli Decl."), App., Ex. 2; Expert Report of Dr. Geoffrey Altshuler ("Altshuler Rep."), Ex. B to Declaration of Dr. Altshuler, App., Ex. 3.; Supplemental Expert Report of Dr. Geoffrey Altshuler ("Supp. Altshuler Rep."), Ex. C to Declaration of Dr. Altshuler, App., Ex. 3. For example, T.J.'s levels of nucleated red blood cells (NRBCs) were pathologically high, revealing reduced uteroplacental flow. NRBCs indicate the fetus is attempting to compensate due to hypoxia. Supp. Altshuler Rep.; Supplemental Expert Report of Dr. John Graham ("Supp. Graham Rep.") at 2, Ex. C to Declaration of Dr. Graham, App., Ex. 1.

T.J. showed signs of cerebral palsy as early as six months of age and eventually was diagnosed with cerebral palsy at eighteen months. Deposition of Rene Junk ("Rene Junk Dep.") at 116-117, 126-27, Ex. A to Declaration of Joseph Eaton ("Eaton Decl."), App., Ex. 4. T.J.'s treating physicians linked his cerebral palsy and resulting neurodevelopmental delay to his premature birth and the large tumor on his umbilical cord. Scialli Rep. at 9.

Chlorpyrifos is one of the most widely used insecticides in the world and it has been studied in both human and animals since 1965. *See, e.g.*, Graham Rep. at 21-23; Schardein & Scialli, The Legislation of Toxicologic Safety Factors: The Food Quality Protection Act with Chlorpyrifos as a Test Case, 13 Reproductive Toxicology 1, 2 (1999), Ex. D to Scialli Decl.,

App., Ex. 2. It is one of a class of pesticides generally known as organophosphates. The potentially toxic effects of organophosphate substances generally, and of chlorpyrifos specifically, are well understood and extensively documented in scientific and medical literature. *See, e.g.*, R.C. Cochran, Appraisal of Risks from Nonoccupational Exposure to Chlorpyrifos, 35 Regulatory Toxicology & Pharmacology 105, 112 (2002) ("Cochran Article") ("[T]he database for chlorpyrifos on reproductive and developmental toxicity is the most complete of any pesticide."), Ex. E to Scialli Decl., App, Ex. 2.

Acute overexposure to organophosphates can cause specific symptoms by affecting specific organs in a specific manner resulting in a well-recognized pattern of "cholinergic" symptoms (meaning symptoms resulting from over stimulation of certain parts of the nervous system). *See* Excerpts from Casarett & Doull's Toxicology (Amdur et al. eds., 4th ed. 1991), Ex. F to Scialli Decl., App., Ex. 2. The well-defined and well-known cholinergic symptoms of an acute overexposure to an organophosphate, such as chlorpyrifos, include lacrimation, salivation, urination, nausea, abdominal pains, diarrhea and pin-point pupils. *Id.* Neither T.J. nor Rene Junk demonstrated any signs and symptoms of acute overexposure. Supplemental Expert Report of Dr. Anthony Scialli ("Supp. Scialli Rep.") at 22, Ex. C to Scialli Decl., App., Ex. 2.

Plaintiff's experts, only one of whom is a medial doctor, contend T.J.'s cerebral palsy and resulting developmental delay were caused by his alleged *in utero* and postnatal exposure to chlorpyrifos. Neither of the Plaintiff's experts can support his or her opinions with any reliable peer-reviewed scientific literature showing an association between chlorpyrifos and premature birth, chorioangioma, cerebral palsy or resulting developmental delay at any exposure level or dose. Plaintiff's experts cannot and have not excluded other potential causes of T.J.'s cerebral palsy and neither Dr. Bearer nor Dr. Abou-Donia utilized an estimate or calculation of T.J.'s

exposure level or dose of chlorpyrifos as a result of a crack and crevice application. Instead, Plaintiff's experts improperly rely upon inapposite animal studies, and pure speculation to reach their conclusions.

C. Plaintiff's experts and their causation opinions.

1. Cynthia Bearer, M.D.

Dr. Bearer is a neonatologist associated with the Rainbow Babies and Children's Hospital in Cleveland, Ohio. Dr. Bearer does not claim to have any experience in toxicology and is not a specialist in the fields of either cerebral palsy or neurodevelopmental delay. Despite this lack of training in relevant fields, Dr. Bearer claims that the rare and extremely large chorioangioma on Rene Junk's umbilical cord is not associated with T.J.'s cerebral palsy and resulting conditions. Dr. Bearer intends to testify that T.J. had no signs of hypoxia at delivery and that "although it may be impossible to quantify precisely the amount of Dursban to which T.J. was exposed, it is more likely than not that the multiple exposures, *in utero* and after birth, caused T.J.'s neurodevelopmental delay." Supplemental Expert Report of Dr. Cynthia Bearer ("Supp. Bearer Rep.") at 2, Ex. B to Eaton Decl., App, Ex. 4.

2. Mohamed Abou-Donia, Ph.D.

Mohamed Abou-Donia, Ph.D. has a doctorate degree in agricultural chemistry and is a research scientist at Duke University Medical Center. Although Dr. Abou-Donia is not a medical doctor, Plaintiff intends to use Dr. Abou-Donia to establish the requisite evidentiary link between T.J.'s alleged exposure to chlorpyrifos and his medical conditions from a toxicological perspective. Dr. Abou-Donia intends to testify that the temporal relationship between T.J.'s alleged exposure to chlorpyrifos and T.J.'s cerebral palsy and resulting neurodevelopmental

delay, along with the known adverse effects of an acute overexposure to chlorpyrifos,[3] establishes that exposure to chlorpyrifos caused T.J.'s medical problems. Despite Dr. Abou-Donia's admission that he does not have accurate exposure data, he further intends to testify that T.J.'s exposure must have been sufficient since T.J. had cerebral palsy and resulting neurodevelopmental delay.

ARGUMENT

I. Standards For Admissibility Of Scientific Evidence Relating To Causation.

A. Plaintiff bears the burden of demonstrating the reliability of her experts' causation methods.

To prove her claim that chlorpyrifos exposure caused T.J.'s cerebral palsy and resulting neurodevelopmental delay, Plaintiff must establish both general and specific causation. *See Nat'l Bank of Commerce (Smits) v. Dow Chem. Co.*, 965 F. Supp. 1490 (E.D. Ark. 1996), *aff'd*, 133 F.3d 1332 (8th Cir. 1998) (excluding experts who attempted to link alleged exposure to chlorpyrifos to birth defects). The first prong of causality in a chemical exposure case, known as "general causation," requires the Plaintiff to prove that the chemical complained of is *capable* of causing the type of injury alleged in humans. *Wright v. Willamette Indus.*, 91 F.3d 1105, 1106 (8th Cir. 1996). If, and only if, the Plaintiff satisfies the first inquiry, the Plaintiff must then

[3] Throughout, DAS is using "chlorpyrifos" as a shortcut for chlorpyrifos and chlorpyrifos-containing products, including Dursban L.O., the specific product applied in the Junk home. In this case, however, there is no appreciable distinction between the two, as Plaintiff's experts have not offered any opinions as to anything other than the insecticidal molecule chlorpyrifos, and indeed, Plaintiff's experts were unaware of the solvents and other inert ingredients in the Dursban L.O. formulation to which Plaintiff was allegedly exposed. *See* Deposition of Mohamed B. Abou-Donia, Ph.D. ("Abou-Donia Dep.") at 88, Ex. C to Eaton Decl., App., Ex. 4 ("Q: You're not rendering any opinions in this case about whether or not the solvents in the Dursban formulation caused any of [the minor T.J.'s] problems? A: Since I don't know the solvents, I cannot tell."); Deposition of Cynthia Bearer, M.D. ("Bearer Dep.") at 169, Ex. D to Eaton Decl., App., Ex. 4 ("Q: You did not review the labels, the warning labels for the Dursban products involved or the material safety data sheets in forming your opinions? A: I did not. Q: Are you aware of the specific Dursban formulation used in the home? A: I am not.").

prove specific causation by demonstrating that the chemical was *in fact* the actual cause of the Plaintiff's alleged injury. *Id.*

Due to the nature of expert testimony and its potential to mislead and confuse the jury, it is necessary for the trial court to initially determine the admissibility of the testimony under Rule 702 before it is presented to the trier of fact. *See, e.g.*, *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 595 (1993). Plaintiff carries the burden of demonstrating to the Court by a preponderance of the evidence that her experts are qualified to render their opinions and that the methodologies underlying their conclusions are scientifically valid. *Id.* at 589-90; *Marmo v. Tyson Fresh Meats*, 457 F.3d 748, 757-58 (8th Cir. 2006). District courts have broad discretion to admit or reject the admissibility of expert testimony. *Peitzmeier v. Hennessy Indus.*, 97 F.3d 293, 296 (8th Cir. 1996).

In reviewing the proffered testimony, the court acts as an evidentiary filter by screening scientific testimony or evidence to ensure its relevance and reliability. *Id*. at 589; *Sappington v. Skyjack, Inc.*, 512 F.3d 440, 448 (8th Cir. 2008). Essentially, the district court acts as a gatekeeper to separate expert testimony based on "good grounds" from "subjective speculation that masquerades as scientific knowledge." *Glastetter v. Novartis Pharm. Corp.*, 252 F.3d 986, 989 (8th Cir. 2001).

The Court may evaluate the reliability of the experts' methods and testimony based on the following list of nonexhaustive and nondispositive *Daubert* factors:

(1) whether the theory or technique can be (and has been) tested;

(2) whether the theory or technique has been subjected to peer review and publication;

(3) the known or potential rate of error; and

(4) whether the theory has been generally accepted in the scientific community.

Smith v. Cangieter, 462 F.3d 920, 923 (8th Cir. 2006) (citing *Daubert*, 509 U.S. at 593-94). Lastly, the Court can consider "whether the expertise was developed for litigation or naturally flowed from the expert's research; whether the proposed expert ruled out other alternative explanations; and whether the proposed expert sufficiently connected the proposed testimony with the facts of the case." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 150 (1999); *Lauzon v. Senco Prod., Inc.*, 270 F.3d 681, 687 (8th Cir. 2001); *see also* Fed. R. Evid. 702 advisory committee notes.

The cornerstone of this Court's review under Rule 702 and *Daubert* is maintaining the standard of reliability. District courts must "make certain that an expert . . . employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire*, 526 U.S. at 141. As noted by the Third Circuit, the Court must assess each step in the expert's analysis:

> [a]ny step [in methodology or reasoning] that renders the analysis unreliable . . . renders the expert's testimony inadmissible. This is true whether the step completely changes a reliable methodology or merely misapplies that methodology.

In re Paoli Yard PCB Litig., 35 F.3d 717, 745 (3d Cir. 1994).

B. The testimony and methods must "fit" the facts of the case and be more than mere speculation.

Even if in applying the above factors a court finds a particular theory to be reliable, testimony regarding that theory is only admissible if it would be helpful to the trier of fact by being sufficiently tied to the facts of the case. *See Daubert*, 509 U.S. at 591. This requires the Court to determine relevancy or "fit" of the testimony. To establish "fit," the "proponent of the testimony must show that the reasoning or methodology in question is applied properly to the facts in issue" and will serve to assist the trier of fact to understand the evidence. *Id.* at 591-93; *Marmo*, 457 F.3d at 758.

Scientific evidence must be more than "subjective belief or unsupported speculation" to be admissible. *Id.*; *Arcoren v. United States*, 929 F.2d 1235, 1239 (8th Cir. 1991). It is well settled in the Eighth Circuit that a court may not admit testimony that "is connected to existing data only by the *ipse dixit* of the expert." *Glastetter*, 252 F.3d at 990 (quoting *GE v. Joiner*, 522 U.S. 136, 146 (1997)). An expert's "bald assurance" that his or her methodology is "scientific" is not sufficient. *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1316 (9th Cir.) ("*Daubert II*") (explaining that "an expert's self-serving assertion that his conclusions were 'derived by the scientific method' [cannot] be deemed conclusive"), *cert. denied*, 516 U.S. 869 (1995). Rather, the proponent of the expert testimony "must show that the expert's findings are based on sound science, and this . . . require[s] some objective, independent validation of the expert's methodology." *Id*.

II. Plaintiff Has Not Met Her Burden Of Proving The Scientific Reliability Of Her Experts' Methodologies For Determining General Causation.

Drs. Bearer and Abou-Donia have presented novel general causation theories using methodologies that lack the support of credible scientific evidence in the form of human epidemiological studies or peer-reviewed, published literature demonstrating *any* link between chlorpyrifos and cerebral palsy or neurodevelopmental delay.

Further, Plaintiff's experts' methods also ignore credible scientific data demonstrating a complete lack of association between chlorpyrifos and cerebral palsy and resulting neurodevelopmental delay and do not satisfy Rule 702 and *Daubert* and its progeny.

A. Plaintiff's experts' methods ignore the fact that there is no credible scientific evidence demonstrating that exposure to chlorpyrifos is capable of causing umbilical cord tumors.

Plaintiff's experts' methods establish that T.J.'s injuries were caused by his alleged exposure to chlorpyrifos and ignore the fact that there is *no* credible, scientific evidence linking exposure to chlorpyrifos to umbilical cord tumors, or cancer.

1. There is no scientific literature linking chlorpyrifos and the rare occurrence of chorioangiomas.

Chorioangiomas are vascular tumors that occur in the placenta and umbilical cord. Large chorioangiomas, such as the one on T.J.'s umbilical cord, are exceedingly rare.[4] *See* Scialli Rep. at 4; Graham Rep. at ¶¶ 51-52.

Although the precise etiology of chorioangiomas is unknown, there is no basis for a conclusion that these vascular tumors are caused by environmental exposures in general or chlorpyrifos in particular. There has been no demonstrated increase in the frequency of chorioangiomas since chlorpyrifos was first manufactured and used in 1965. Graham Rep. at ¶ 48. In fact, in previous studies concerning the occurrence of these types of tumors, the vast majority of clinical cases occurred prior to the introduction of chlorpyrifos into the market in 1965. *Id.* The dearth of any published literature or scientific studies attributing vascular tumors to chlorpyrifos exposure during pregnancy demonstrates that there is no basis for any causal association between chlorpyrifos and chorioangiomas.

[4] The chorioangioma, along with T.J.'s prematurity, is the most likely cause of T.J.'s injuries. Plaintiff's experts have failed to consider this primary cause of T.J.'s injuries. *See infra* Part III.B.

2. Plaintiff's experts' methods ignore the fact that chlorpyrifos is not a carcinogen and is not capable of causing umbilical cord tumors.

Chorioangioma is a form of tumor, yet Plaintiff's experts wholly failed to consider the objective scientific evidence that chlorpyrifos is simply not carcinogenic. Not only is there *no* affirmative evidence linking chlorpyrifos to tumors or cancer, there is ample evidence *disproving* the notion that chlorpyrifos can cause a tumor or cancer. For example, the Health Effects Division ("HED") of the Environmental Protection Agency ("EPA") stated, "[C]hlorpyrifos was evaluated for carcinogenic potential in both rats (2 studies), and mice (2 studies). There was no evidence of treatment-related tumors or carcinogenicity." *See* Toxicology Chapter for Chlorpyrifos, May 6, 1999, at 5, Ex. G to Scialli Decl.

Because chlorpyrifos is not carcinogenic, there is simply no basis for Plaintiff's experts' speculative theory that exposure to chlorpyrifos could cause the growth of a rare chorioangioma or any other similar tumor.

3. Plaintiff's experts admit that there is no credible scientific literature linking chlorpyrifos and Rene Junk's chorioangioma.

Drs. Bearer and Abou-Donia largely concede that there is *no* link between T.J.'s alleged exposure to chlorpyrifos and the large chorioangioma on his umbilical cord. Dr. Bearer confirmed that chorioangiomas are a rare condition and that this was the first case in which she had evaluated a chorioangioma. Bearer Dep. at 94. She willingly admitted that there is no scientific evidence to support the proposition that chlorpyrifos is capable of causing chorioangiomas. *Id*. at 118, 133. Likewise, Dr. Abou-Donia conceded that he was unaware of any published literature linking chlorpyrifos with chorioangiomas. Abou-Donia Dep. at 71. As a toxicologist, Dr. Abou-Donia has neither conducted any independent research nor been involved in any *in vivo* or *in vitro* studies on chorioangiomas. *Id.* at 59. Dr. Abou-Donia further

confirmed that he was *not* opining as to whether exposure to chlorpyrifos caused Rene Junk's chorioangioma. *Id*. at 89, 122.

B. Plaintiff's experts' methods ignore that there is no credible scientific evidence demonstrating that exposure to chlorpyrifos is capable of causing cerebral palsy and resulting neurodevelopmental delay.

1. Cerebral palsy is a nondegenerative and nonprogressive disability with prematurity as the most common identified cause.

Cerebral palsy is a relatively common, nonprogressive, nondegenerative developmental disability that is generally characterized by abnormal muscle tone, posture, coordination and reflexes. Graham Rep. at ¶ 30; *see also* Bearer Dep. at 112. The majority of children with cerebral palsy present with symptoms as infants and toddlers, and the diagnosis of cerebral palsy is made before age 2 years. Cerebral palsy is not the result of a recognized progressive or degenerative brain disease. Supp. Graham Rep. at 1. Cerebral palsy occurs in about 1 in 500 live births, resulting in about 8500 new cases of cerebral palsy each year. Scialli Rep. at 3. The most common identified cause of cerebral palsy is prematurity. *Id.* at 4. Cerebral palsy and developmental delay can be caused by decreased oxygen delay to the fetal brain. *Id.* at 3.

2. There is no credible scientific literature of any kind linking cerebral palsy to chlorpyrifos.

There is no evidence demonstrating that cerebral palsy and resulting neurodevelopmental delay are causally associated with environmental agents in general, or chlorpyrifos in particular. *See* Graham Rep. at ¶¶ 34, 38. And Plaintiff's experts fail to cite any such literature. This Court should consider the absence of medical literature supporting the conclusions of Drs. Bearer and Abou-Donia in assessing the reliability of their testimony. *Nat'l Bank of Commerce v. Associated Milk Producers, Inc.*, 191 F.3d 858, 864 (8th Cir. 1999) (highlighting the lack of scientific studies in a chemical exposure case and holding that "the existence of such studies . . .

is not a sine qua non for imposing liability on defendant; but as [*Daubert*] also makes clear, they are a factor"); *cf*. *Turner v. Iowa Fire Equip Co.*, 229 F.3d 1202, 1208-09 (8th Cir. 2000).[5] Plaintiff's experts cannot identify **any** published literature showing a scientific basis for the conclusion that exposure to chlorpyrifos *in utero* or postnatally is capable of causing cerebral palsy. Dr. Abou-Donia admitted that the studies that he purports to rely on do not specifically reference a link between cerebral palsy and chlorpyrifos exposure. Abou-Donia Dep. at 77. Likewise, Dr. Bearer admitted that she was *not* "aware of any published peer reviewed literature linking chlorpyrifos exposure to the diagnosis of cerebral palsy." Bearer Dep. at 132.

3. There is no secular trend data or analysis demonstrating an association between chlorpyrifos and cerebral palsy.

Insecticidal products with chlorpyrifos as their active ingredient have been widely used, to varying degrees of intensity, since the introduction of chlorpyrifos in 1965. *See* Graham Rep. at 21-23; Supp. Graham Rep. at 3; *see also* Deposition of William Chen, Ph.D "(Chen Dep.") at 13, Ex. E to Eaton Decl.; Interpretation of Chlorpyrifos Exposure Incident Data, Dec. 9, 1994, Ex. 94 to Chen Dep., Ex. F to Eaton Decl.; Analysis of Insecticide Applications by Product Use Patterns and Chemical Classification, June 13, 1996, Ex. G to Eaton Decl. If Drs. Bearer and Abou-Donia's methods were reliable, they would be able to demonstrate that the incidence of cerebral palsy and resulting neurodevelopmental delay has increased in populations exposed to chlorpyrifos using secular trend analysis. Conversely, if Plaintiff's experts' causal assumptions

[5] In *Turner*, the Eighth Circuit noted that a finding of reliability does not *necessarily* require that an expert always cite published studies to establish general causation. *Turner*, 229 F.3d at 1208-09 (8th Cir. 2000) (citing *Heller v. Shaw Indus.*, 167 F.3d 146, 155 (3d Cir. 1999)). However, *Turner* confirms that admissibility is contingent on a qualified *medical* expert establishing causation by eliminating all other possible causes of a plaintiff's condition other than the chemical substance and by relying on a vast clinical experience and/or a clear, unmistakable temporal link between exposure and illness. *Id*. at 1209. Plaintiff's experts do not satisfy any of these criteria.

were correct they would be able to show that the rate of cerebral palsy and neurodevelopmental delay has decreased in populations that are no longer exposed to chlorpyrifos or were not exposed to chlorpyrifos as frequently as other populations.

Since the first use of chlorpyrifos in 1965 there has been no demonstrated increase in cases of cerebral palsy. Graham Rep. at 23. And there have been no studies to support a contention that the incidence of cerebral palsy and neurodevelopmental delay has decreased in populations where the exposure to chlorpyrifos has decreased significantly. *Id.*; *see also* Supp. Graham Rep. at 4. Simply put, there is no evidence showing that children exposed to chlorpyrifos have an increased risk of cerebral palsy or neurodevelopmental delay. Scialli Rep. at 7. Drs. Bearer and Abou-Donia cannot show any appreciable increase or decrease in the rate of either condition connected with exposure to chlorpyrifos.

4. Plaintiff's experts' methods consciously ignore the weight of published literature confirming the lack of a causal connection between chlorpyrifos exposure and cerebral palsy and resulting neurodevelopmental delay.

Plaintiff's experts methods not only ignore the body of literature relating to cerebral palsy and resulting neurodevelopmental delay, they also ignore the vast body of literature relating to chlorpyrifos generally. In so doing, Plaintiff's experts "reason from an end result in order to hypothesize what needed to be known but was not." *Sorensen v. Shaklee Corp.*, 31 F.3d 638, 649 (8th Cir. 1994). What *is* known is that chlorpyrifos is one of the most widely studied insecticides in the world. What is *not* known is any link in those studies between exposure to chlorpyrifos and cerebral palsy and resulting neurodevelopmental delay. By using methodologies that reasoned from what was *not known* (but needed to be), Plaintiff's experts' methodologies are unreliable and should be excluded.

The data for chlorpyrifos on reproductive and developmental toxicity is the most complete of any pesticide. In evaluating the admissibility of Plaintiff's experts' testimony, the Court may note the body of neutral, peer-reviewed literature citing scientific studies showing that exposure to chlorpyrifos after residential crack and crevice applications does not cause birth injuries in humans. Dr. James Schardein and Dr. Anthony Scialli, board certified in obstetrics and gynecology and a reproductive toxicologist who also is a testimonial expert for DAS here, published in 2002 a comprehensive review of reproductive and developmental toxicity literature relating to chlorpyrifos. The authors concluded that there is no significant toxicity in the young compared with the adult and there is no evidence of specific toxicity of chlorpyrifos for the developing central nervous system. Schardein & Scialli, The Legislation of Toxicological Safety Factors: The Food Quality Protection Act With Chlorpyrifos As A Test Case, 13 Reproductive Toxicology 1 (1999), Ex. D to Scialli Decl.

In addition, R.C. Cochran, a toxicologist with the California Department of Pesticide Regulation, published a chlorpyrifos review article in a peer-reviewed journal. Cochran Article, Ex. E to Scialli Decl. Cochran examined illness report data for nonoccupational exposure to chlorpyrifos and found that 77 percent of incidents occurred because of reentry into a treated area within one hour of treatment. Cochran then concluded that the likely cause of the reported symptoms was due to the unpleasant odors associated with Dursban. *Id.* at 106. Cochran noted that developmental toxicity from exposure to chlorpyrifos did not occur at levels below maternal toxicity, which means that the only way that a rat pup can show any signs of illness is if his mother is significantly ill. *Id.* at 105. Based on the data complied from animal studies, Cochran concluded that:

> [t]here is insufficient evidence that human infants are more susceptible to the toxicity of chlorpyrifos than adults and small children, and there is no compelling

> evidence that chlorpyrifos causes any developmental neurotoxicity under physiological relevant conditions. Consequently, there do not appear to be data which warrant using an extra safety factor to take into account potential pre- and postnatal developmental toxicity.

Id. at 112. Accordingly, the risks of chlorpyrifos exposure based on data extrapolation "have been overstated by more than a 1000-fold." *Id.* Analyzing the extant data, the study found that label approved uses of chlorpyrifos did not constitute a significant health risk to the general public. *Id.* at 116.

Plaintiff's experts' methodologies ignore this scientific and medical literature discounting their conclusions. An expert's failure to consider relevant literature is grounds for exclusion. *See, e.g., Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202 (4th Cir. 2001).

5. Plaintiff's experts' methods ignore literature linking chorioangioma and cerebral palsy.

Dr. Bearer testified that she does not believe the chorioangioma had any effect on T.J. during Rene Junk's pregnancy. Bearer Dep. at 135-136. Contrary to Dr. Bearer's opinion, a large chorioangioma like the one on T.J.'s umbilical cord has been associated with cerebral palsy in an infant at 31 weeks gestation, who experienced enlarged ventricles and a diagnosis of cerebral palsy at one year of age. Supp. Graham Rep. at 2-3; *see also* Hiarigaya, A., et al., Premature infant with severe periventricular leukomalacia associated with a large placental chorioangioma: a case report. 22 J. Perinatology 252 (2002), Ex. O, Scialli Decl. The chorioangioma on T.J.'s umbilical cord was even larger than the one in this publication. *See* Graham Rep. at ¶ 49. Dr. Bearer consciously ignored or was unaware that chorioangioma has been linked to cerebral palsy.

C. Plaintiff's experts improperly extrapolate animal data to attempt to establish that chlorpyrifos causes cerebral palsy and resulting neurodevelopmental delay.

Because Plaintiff's experts have no scientifically reliable evidence linking exposure to chlorpyrifos to cerebral palsy and resulting neurodevelopmental delay, Plaintiff's experts improperly extrapolate from irrelevant and ill-fitting animal studies. This methodology is unreliable and should be rejected.

1. Human data is required for conclusions that there is a causal relationship between a chemical exposure and an outcome in humans.

To support their proffered causation testimony, Drs. Bearer and Abou-Donia principally rely on the extrapolation of data from animal studies, both *in vivo* (whole animal studies) and *in vitro* (cellular level studies). Dr. Bearer cites various studies using rats to form her conclusion that "prenatal/postnatal exposure to chlorpyrifos is associated with neurodevelopmental delay both in animals and humans." Expert Report of Dr. Cynthia Bearer ("Bearer Rep."), Ex. H to Eaton Decl. Dr. Bearer goes even further by testifying that even in the absence of any human data, a causal link can be made through animal studies to greater than fifty (50) percent scientific certainty. Bearer Dep. at 108. Similarly, Dr. Abou-Donia cites his study of brain neuronal cell death in the cerebellum of rat offspring to bolster his conclusion that repeated exposure to chlorpyrifos can result in chronic neurotoxicity.[6]

In determining causation, it is necessary to follow the principle that human data is required for conclusions that there is a causal relationship between an exposure and an outcome

[6] As will be discussed, Dr. Abou-Donia's study used *in utero* exposure at a rate of 1.0 mg/kg/d of chlorpyrifos, which is substantially higher than the actual level human exposure.

in humans.[7] Scialli Rep. at 6. Courts across the country have affirmed this principle in holding that animal studies are not a valid basis for extrapolating conclusions about human disease causation without a reliable scientific link showing that the effect on animals can predict an effect in humans. By way of example, in *Goewey v. U.S.*, 886 F. Supp. 1268 (D.S.C. 1995), *aff'd*, 106 F.3d 390 (4th Cir. 1997) (table, text at 1997 WL 35348), *cert. denied sub nom. Goewey v. Fluor Daniel, Inc.*, 522 U.S. 1045 (1998), the Court excluded Dr. Abou-Donia's proffered testimony in finding "no scientific credibility in the test of [the plaintiff's] blood performed for the purposes of detecting phosphorylated neurofilaments." 866 F. Supp. at 1280. Further:

> Again, Dr. Abou-Donia's conclusions are extrapolated from his work on chickens. Because the test has never been performed on a human before, has not been published or subject to peer review, and had been criticized by another one of Plaintiff's experts . . . it cannot meet the threshold of reliability under *Daubert* and therefore, may not be probative of causation.

Id.

2. Plaintiff's experts' unscientific methodologies rely on improper extrapolation from animal studies.

Animal studies are sometimes useful in the study of teratogenics in that they may provide a means for identifying chemicals with disease causing potential. Graham Rep. at ¶ 45. However, animal studies "are not and should not be considered to represent adequate proof that an agent is teratogenic in humans." *Id.* Because of the vast differences among species, particularly in the areas of placental development, metabolism and embryonic development, it is

[7] It should be noted preliminarily that the determination of causation in a lawsuit is not the same as a regulatory determination of a protective level of exposure. *See Glastetter*, 252 F.3d at 991 ("The FDA's 1994 decision that Parlodel can cause strokes is unreliable proof of medical causation in the present case because the FDA employs a reduced [causation] standard . . . . "); *see also* Dow AgroSciences' Brief in Support of its Motion in Limine to Exclude Evidence or Argument Relating to the June 2000 Memorandum of Agreement with EPA [Docket No. 121].

inappropriate to directly extrapolate findings in animals to humans for purposes of causation. *Id*. Although it may be possible to establish general causation for humans indirectly through the use of animal data, this can only be accomplished through the use of scientific evidence that establishes "precise qualitative and quantitative relationships between measurements made in the test species and production of the particular disease in humans." Expert Report of Dr. Christopher Borgert ("Borgert Rep."), Ex. B to Declaration of Dr. Borgert, App., Ex. 5.

Dr. Bearer confirmed the differences between species that limit extrapolation from animal data to humans:

> Animals don't read, so you can't really tell if the reading ability of a child is going to be affected by some of the things we see in animals. . . . I think sometimes there are interspecies differences, that is where the ten fold safety factor comes in. Probably having to do with differences in metabolism.

Bearer Dep. at 110-111. In *Glastetter v. Novartis Pharm. Corp.*, 252 F.3d 986 (8th Cir. 2001), the Eighth Circuit confirmed that animal studies were insufficient to prove causation without scientific evidence that the results can be extrapolated to effects in humans. *Id*. at 989; *see also Daubert II*, 43 F.3d at 1320 (the admissibility of animal studies depends on the existence of "some authority for extrapolating human causation from teratogenicity in animals."); *Rider v. Sandoz Pharms.*, 295 F.3d 1194, 1202 (11th Cir. 2002) (excluding testimony based on animal studies because plaintiffs failed to offer a rationale for the conclusion that effects in animals may be extrapolated to humans); *Sanderson v. Int'l Flavors & Fragrances, Inc.*, 950 F. Supp. 981, 997 (C.D. Cal. 1996) ("In order for animal studies to be admissible to prove causation in humans, there must be good grounds to extrapolate from animals to humans.").

Plaintiff's experts offer no scientific evidence to support their methodologies of extrapolating animal data to human outcomes. And the data that they do cite does not measure up to the standard of "precise qualitative and quantitative relationships" necessary to indirectly

establish a causal link between a chemical exposure and an injury through animal studies. Reliance on animal testing alone does not meet the level of reliability required by *Daubert*.

Even assuming Plaintiff's experts were able to prove that their methodologies for extrapolating animal data could demonstrate an indirect association between chlorpyrifos exposure and a specific injury, there is no evidence to show that chlorpyrifos is capable of producing either cerebral palsy or neurodevelopmental delay in an animal model. Borgert Rep. at 3.

(a) There is no animal model demonstrating a link between exposure to chlorpyrifos and cerebral palsy and neurodevelopmental delay.

None of the studies cited by Plaintiff's experts demonstrate that chlorpyrifos causes cerebral palsy and neurodevelopmental delay. Borgert Rep. at 3. In fact, the literature relied upon by Plaintiff's expert has nothing to do with either condition because a validated animal model that can reliably predict the result of cerebral palsy and developmental delay in humans does not exist.

Due to the unknown etiology of cerebral palsy and resulting neurodevelopmental delay, and the lack of a validated animal model for human predictivity, it is impossible for Plaintiff's experts to extrapolate the vast amount of neurotoxicological and developmental toxicological research performed on animals with chlorpyrifos to infer causation in humans. *See id.* Because of the absence of direct human and indirect animal evidence linking chlorpyrifos with either cerebral palsy or neurodevelopmental delay, Drs. Bearer and Abou-Donia lack any scientifically valid method to support their conclusions.

(b) In vivo animal studies use huge doses of chlorpyrifos that are maternally toxic and are not suggestive of causation in humans.

The animal studies cited by Drs. Bearer and Abou-Donia use massive doses of chlorpyrifos to produce maternal toxicity and fetal effects.[8] Graham Rep. at ¶ 45. As noted by Dr. Graham, "[e]xperiments in animals often employ dosages that are many times greater than those likely to occur in humans, and toxic effects to the animal mother being dosed may impact the fetus and confound the interpretation of fetal outcome." *Id.* The reliability of Plaintiff's experts' methods is further diminished because when an animal mother becomes ill because of exposure to massive doses of a chemical agent it is difficult to interpret whether any outcome in the fetus is due to the chemical exposure or to the mother's illness. *Id.* As demonstrated in the peer-reviewed, scientific literature, chlorpyrifos has minimal evidence of neurotoxicity in humans. In the limited case reports that have reported neurotoxic effects, the neurotoxicity occurs only after extremely high doses resulting in significant systemic effects and patient compromise. Expert Report of Dr. Michael Ross ("Ross Rep."), Ex. B to Declaration of Dr. Ross, App, Ex. 6.

Plaintiff's experts rely on studies that employ massive levels of chlorpyrifos to produce the effects on which their data is based. Even the "very small doses" of chlorpyrifos referenced by Dr. Abou-Donia (1.0 mg/kg/day) were thousands of times greater than what a human is typically exposed to in standard crack and crevice applications. Abou-Donia Dep. at 6; *see also*

[8] The doses used in the animal studies cited by Drs. Bearer and Abou-Donia are well above any no observable effect level, or NOEL. Even when adding a largely unnecessary 100x "safety factor" to "account for" species differences and exposure to children and infants, residential crack and crevice applications do not approach levels of exposure or doses that produce adverse affects in animals. *See infra* Part III.C (discussing exposure, dose, and dose/response).

Cochran Article at 21, Ex. E to Scialli Decl. Because the studies referenced by Drs. Bearer and Abou-Donia employed chemical doses at levels much higher than that experienced by humans, the data cannot be used to provide useful information by which to evaluate a possible causation proposition in humans. Scialli Rep. at 7. Any opinions formed from this data are purely theoretical and should therefore be excluded as inherently unreliable.

(c) Subcutaneous exposure pathways employed in animal studies are improper to determine human exposure.

There is a basic scientific question as to "whether a model system, which relies on nearly lethal intraperitoneal and subcutaneous injections of chlorpyrifos into newborn rats, is physiologically relevant to the developing nervous system of a human fetus *in utero.*" Cochran Article at 111, Ex. E to Scialli Decl. The animal studies cited by Drs. Bearer and Abou-Donia authored by Dr. Theodore Slotkin's laboratory employ nearly lethal doses of chlorpyrifos that are diluted in dimethylsulfoxide (DMSO) and injected subcutaneously in the fat underneath the skin of the rats. This method of administration results in rapid absorption of chlorpyrifos into the blood in high peak concentrations. Scialli Rep. at 17. These methods of chlorpyrifos administration, while capable of producing a number of effects in rat fetuses, have absolutely no correlation to human exposure methods and thus cannot be used to extrapolate effects in human fetuses. This type of subcutaneous route of exposure is improper since chlorpyrifos exposure in the human population "does not occur through subcutaneous injection." Brent & Weitzman, The Current State of Knowledge About the Effects, Risks and Science of Children of Environmental Exposures, 113 Pediatrics 1158, 1160 (2004); Ex. H to Scialli Decl. Many of the animal studies on chlorpyrifos from Dr. Slotkin's lab were "difficult to apply" to humans because the exposures that were used were "very high and were administered subcutaneously." *Id.* at 1162.

In determining the reliability of Plaintiff's experts' methodologies of extrapolation, the Court should consider the Plaintiff's experts' failure to account for the difference in exposure pathways in concluding that data from rats subcutaneously injected with high doses of chlorpyrifos can be extrapolated to predict causal effects in humans at much lower levels in much less invasive administration methods. Dr. Bearer in fact admitted that she failed to make any formal correlation between the exposure scenarios in the animal studies she cited and the level of exposure in the Junk home. Bearer Dep. at 146-147. Because Plaintiff's experts made no attempt to correlate the exposure pathways and dosage levels in the animal studies with that of typical human exposures in developing their opinions, the Court should exclude their methodologies as inappropriate for human comparison.

(d) Dr. Slotkin admits his studies are novel and untested.

As noted, Dr. Bearer relies or at least references numerous *in vitro* and *in vivo* studies conducted by Theodore Slotkin, Ph.D. Dr. Slotkin does not utilize appropriate routes of exposure and/or dose levels even remotely similar to what one would expect after a residential crack and crevice application. Supp. Scialli Rep. at 14-16. Accordingly, his studies have been largely rejected by the scientific community. More importantly, Dr. Slotkin has admitted outside of the courtroom to other academicians that his publications and research on chlorpyrifos are **novel, untested theories** driven by grant proposals and funding:

> For academic scientists, the primary emphasis is on novelty: novelty of the compound studied, novelty of the approach taken, novelty of the technology employed, novelty of a basic biological mechanism that is responsible for the effect. Essentially, a successful project produces publications in the top journals in the field, obtains funding, and equally important, opens new doors and establishes new approaches that will guarantee continued funding in future years.

T.A. Slotkin, Guidelines for Developmental Neurotoxicity and Their Impact on Organophosphate Pesticides: A Personal View from an Academic Perspective, 25 NeuroToxicology 631, 633 (2004), Ex. I to Eaton Decl.

Again, none of the Slotkin studies cited by Dr. Bearer demonstrate that chlorpyrifos is capable of causing chorioangiomas, cerebral palsy and resulting neurodevelopmental delay based on residential crack and crevice application as occurred in this case.

3. The human literature relied on by Plaintiff's experts does not support a general causation link between chlorpyrifos exposure and cerebral palsy and resulting neurodevelopmental delay.

Plaintiff's experts make gigantic and speculative leaps in assuming that if chlorpyrifos is capable of producing *X* effects, it must be capable of causing *Y* results. Drs. Bearer and Abou-Donia cite to various published human studies, which do not evaluate cerebral palsy or neurodevelopmental delay, in support of their speculative theory that chlorpyrifos exposure is capable of causing such conditions. None of the studies support Plaintiff's experts' methods and opinions.

(a) The Berkowitz Study.

In both her deposition and her expert reports, Dr. Bearer cites several published studies to support her contention that "prenatal/postnatal exposure to chlorpyrifos is associated with neurodevelopmental delay both in animals and humans" including the Berkowitz study. Bearer Rep.; Supp. Bearer Rep. (citing Berkowitz GS, et al., In Utero Pesticide Exposure, Maternal Paraoxonase Activity, & Head Circumference, 112 Envtl. Health Persp. 388 (2004)). The Berkowitz study evaluated the relationship between the urinary concentrations of the metabolic 3,5,6-trichloro-2-pyridinol (3,5,6 TCP) that measured chlorpyrifos exposure in pregnant women and the head circumference of their children. The Berkowitz study confirms the level of exposure to chlorpyrifos is not determinative of head circumference. There was no significant

association between birth weight or length of head circumference and 3,5,6 TCP, the primary metabolite of chlorpyrifos. Supp. Scialli Rep. at 59. Beyond the lack of causal correlation between head circumference and exposure, the study did not purport to show any association between chlorpyrifos and cerebral palsy or neurodevelopmental delay. *Id.* at 12. At no time does Dr. Bearer move beyond her bald assertion that the literature in some manner supports her method or theory in **this** case. Dr. Bearer does not attempt to articulate for the Court the causal link between the cited literature and her methodologies. For example, Dr. Bearer concludes that since small head circumference is associated with poor neurodevelopmental outcomes, that T.J. must have had small brain matter as a result of his alleged exposure to chlorpyrifos despite the fact that his head circumference was *normal* for his premature gestational age. Bearer Dep. at 36. None of the literature that she cites supports her scientifically unreliable theory.

(b) The Whyatt Study.

Dr. Abou-Donia relies on a study by Whyatt et al., which reported an association between umbilical cord plasma chlorpyrifos levels and fetal birth weight decreases among minority women living in New York City during pregnancy. *See* Expert Report of Dr. Mohamed Abou-Donia ("Abou-Donia Rep."), Ex. J to Eaton Decl. (citing Whyatt RM, et al., Prenatal Insecticide Exposures and Birth Weight and Length Among An Urban Minority Cohort, 112 Envtl. Health Persp. 1125 (2004)). Reliance on this study by Plaintiff's experts is misplaced because the tentative association between birth weight and chlorpyrifos exposure was found to be inconsistent with findings from other studies. *See* Graham Rep. at 19. Prenatal growth restriction is "more common in populations that are of lower socioeconomic status, African-American, teenagers, unmarried, and cigarette smokers." *Id.* at 27. Additionally, younger mothers are likely to be smaller in size and pregnant for the first time, facts that increase their likelihood for constraint-related decreases in size at birth. *Id.* at 27-28. Further, the vast majority

of syndromes that result in fetal growth deficiency, many of which are genetic in nature, are unrelated in any way to chemical exposure. *See* Graham Rep. at ¶ 16. Here, T.J.'s birth weight was consistent with his premature gestational age. *See* Scialli Rep. at 3 (discussing how T.J.'s birth weight of 2260g was above the 90th percentile for infants born at 31-32 weeks gestation). Therefore, the findings on the Whyatt study are not applicable.

D. Chlorpyrifos is not a teratogen and is not capable of causing pre-birth abnormalities.

Plaintiff's experts contend *in utero* exposure to chlorpyrifos caused T.J.'s cerebral palsy and resulting neurologic impairment. Teratogenicity refers to the causation of birth defects (or, more clinically, permanent abnormalities of structure or function in an organism exposed during embryonic or fetal life). *See* Graham Rep. at ¶ 24.

The cause of most birth defects is unknown, although many birth defects are caused by a random gene mutation not linked to any chemical agent. *Id.* at ¶ 16, 20. To establish a particular chemical as a human teratogen, a specific scientific protocol and methodology must be followed, including analysis of epidemiological studies, secular trend data, animal models, dose-response curves, and biological plausibility. Graham Rep. at 27, 32. Chlorpyrifos has never been classified as a teratogen, and any reliable methodology to determine whether a substance is teratogenic would likewise fail to identify chlorpyrifos as a teratogen.

There is no credible scientific evidence that chlorpyrifos is a teratogen, and thus capable of causing a pre-birth injury like cerebral palsy and resulting neurodevelopmental delay.

Dr. Bearer ultimately concluded in her deposition that chlorpyrifos does not cause birth defects or major malformations. Bearer Dep. at 153-54.

E. Plaintiff's experts' general causation methods fail to satisfy the *Daubert* factors.

Using the *Daubert* factors as a guide, the speculative methods and opinions of Drs. Bearer and Abou-Donia fail to pass muster under the reliability standard of admissibility. As explained above, there is no support for the conclusion that chlorpyrifos has any causal connection to either cerebral palsy or neurodevelopmental delay.

1. The methods have not been tested.

The first factor articulated by *Daubert* is whether the theory can be (and has been) tested. *Daubert*, 509 U.S. at 593-94. Drs. Bearer and Abou-Donia have no evidence that exposure to chlorpyrifos is capable of causing chorioangioma, cerebral palsy or neurodevelopmental delay. Even though Plaintiff's experts rely heavily on *in vivo* and *in utero* animal studies, they cannot cite to any animal model that demonstrates their theory has been tested and replicated.

2. The methods have not been subject to peer review.

Second, the Court should also examine whether the Plaintiff's experts' methods have been subjected to peer review scrutiny and publication. *Id*. Here, Plaintiff's experts' unscientific methodologies lead them to conclusions that are inconsistent with, and undercut by, the extant peer-reviewed, published medical and scientific literature relating to chlorpyrifos.

3. The methods are not generally accepted.

The Court should also take into account whether the methods and opinions of Drs. Bearer and Abou-Donia have been generally accepted in the scientific community. *Id.* Plaintiff's experts have offered no evidence of general acceptance and, as DAS has shown, there is no evidence that the scientific community has accepted the conclusion that there is any connection between cerebral palsy or neurodevelopmental delay and exposure to organophosphates generally or chlorpyrifos in particular.

Evaluation of these factors, along with the wealth of information concerning chlorpyrifos that is available to the Court, demonstrate that Plaintiff's experts' testimony should be excluded because it is inherently speculative, untestable, and subject to unknowable and potentially infinite error rates. This is precisely the type of expert evidence that the *Daubert* court sought to prevent.

4. Plaintiff's experts' methods are litigation-driven.

Another factor courts may consider in assessing the reliability of the experts' methods and opinions is whether the experts' conclusions flow naturally from their research, or were developed solely for the purpose of litigation. *See* Fed. R. Evid. 702 advisory committee notes; *Daubert II* at 1316.

Dr. Bearer's conclusions in this case do not flow from her research, as she has *never* studied chlorpyrifos in her lab. *See* Bearer Dep. at 48:

Q. Have you conducted any human studies relating to chlorpyrifos?

A. No.

Q. Have you conducted any human studies relating to organophosphate pesticides?

A. No.

Q. Have you conducted any bench work or lab work relating to chlorpyrifos?

A. No.

Q. Have you conducted any bench work or lab work related to organophosphate pesticides?

A. No.

Dr. Bearer's opinions were formed *solely* for the purposes of this litigation, which is a factor that weighs in favor of excluding her testimony.

As part of her academic research, Dr. Bearer did write a textbook chapter on the effect of environmental agents on a fetus. This chapter lists some twenty-six (26) environmental agents that cause developmental effects, but chlorpyrifos is not one of them. C.F. Bearer, *Occupational*

and Environmental Risks to the Fetus, in NEONATAL-PERINATAL MEDICINE (Fanaroff & Martin, eds., 7th ed. 2001) 188, 192, Ex. K to Eaton Decl. Dr. Bearer also noted that "[i]t is very challenging for the neonatologist confronted with a neonate who may have a problem as a result of an environmental exposure to determine the cause of that problem. Confirming that etiologic agent is almost impossible and requires several steps." *Id.* at 197. Dr. Bearer goes on to outline the steps involved, from obtaining a "detailed exposure history" to reviewing hotlines with epidemiological information about previous outcomes and associations with exposure. *Id.*

Likewise, on Dr. Bearer's homepage for her position at Case Western Reserve University, she mentions that "[c]erebral palsy (CP) is one of the major neurodevelopmental handicaps of premature infants, *yet its etiology remains poorly understood.*" Bearer website, http://neurowww.cwru.edu/faculty/bearer/index, Ex L to Eaton Decl. (emphasis added). Although Dr. Bearer uses this methodology in her academic and clinical practice, she wholly abandons it for the purposes of this litigation, determining that exposure to chlorpyrifos can cause cerebral palsy and resulting neurodevelopmental delay (and also determining, without conducting a detailed exposure history, that Rene Junk's and T.J.'s alleged exposure were sufficient to in fact cause T.J.'s injuries, *see infra* Part III.C).

Likewise, Dr. Abou-Donia has formulated methods and opinions solely for the purposes of this litigation. He admits that he follows the generally accepted criteria for determining causation (called the Bradford Hill factors) in his teaching and academic endeavors. *See* Abou-Donia Dep. at 39. Yet, within his litigation consulting work, he started utilizing terms such as "general causation" and "specific causation" to follow the dictates of the legal field, as opposed to the standard Bradford Hill methodology. *See id.* at 43. Dr. Abou-Donia's methodology for

determining causation is *precisely* dictated by litigation, another factor that weighs in favor of exclusion.

F. Plaintiff's experts' methods have been deemed unreliable and excluded in similar cases.

It is worthwhile to note that this Court will not be forging new territory in assessing the reliability (or unreliability) of the experts' methodologies. At least two other courts have already rejected causation theories similar to the ones espoused here by Drs. Bearer and Abou-Donia and have excluded their proffered testimony.

In *Kerns v. Hobart Brothers Co.*, an unpublished state court decision from Ohio, the court excluded Dr. Bearer from testifying that exposure to certain chemicals *in utero* were capable of causing birth defects and that those chemicals did in fact specifically cause such defects. *Kerns vs. Hobart Bros. Co.*, Case No. 05-235 (Miami County, Ohio Oct. 3, 2007), Order Granting Defendants' Motion to Exclude the Testimony of Dr. Holland and Dr. Cynthia Bearer, Ex. M to Eaton Decl. Dr. Bearer could point to no scientific literature showing that the chemicals were capable of causing birth defects in humans. She then attempted to make the unsubstantiated leap to causation by pointing to animal studies that had no correlation to the potential causal effect in humans. The *Kerns* court held that there was "simply too great an analytical gap between the data and the opinions proffered" and excluded Dr. Bearer's testimony as unsound and unreliable. *Id.* at 4.

Similarly, the Fourth Circuit affirmed a decision from a South Carolina district court, excluding Dr. Abou-Donia's testimony regarding causation in a chemical exposure case. *Goewey v. United States*, 886 F. Supp. 1268 (D.S.C. 1995), *aff'd*, 106 F.3d 390 (4th Cir. 1997). The district court held that Dr. Abou-Donia's testimony did not meet the threshold test of reliability contemplated by *Daubert* because, as in this case, Dr. Abou-Donia had improperly

extrapolated animal data to reach to the unsupported conclusion that exposure to a chemical can cause a particular result in humans. *Goewey*, 886 F. Supp. at 1280.

III. Plaintiff's Experts Lack Scientifically Reliable Methodologies For Determining Specific Causation.

Any specific causation determination starts with the assumption that general causation is proven. Because Plaintiff's experts' methods and opinions as to general causation utilize a methodology that is unreliable and inherently speculative, the Court need go no further. Accordingly, expert testimony as to specific causation must be excluded, and summary judgment granted in favor of the defendant, if there is no reliable expert testimony as to general causation. *Raynor v. Merrell Pharms. Inc*., 104 F.3d 1371, 1376 (D.C. Cir. 1997) (holding that if a "plaintiff is not able to establish general causation, it is unnecessary to consider whether the plaintiff can establish specific causation."); *see also, e.g.*, *Grant v. Pharmative, LLC*, 452 F. Supp. 2d 903, 910 (D. Neb. 2006) (excluding medical doctor's specific causation opinion as "scientifically invalid because general causation had not been proved"); *Hollander v. Sandoz Pharms. Corp.*, 95 F. Supp. 2d 1230, 1239 n.27 (W.D. Okla. 2000) ("Because the court finds the plaintiffs have failed to establish general causation, it is unnecessary to address in detail the defendant's argument that they are also unable to demonstrate specific causation.").

Even assuming the Court reaches Plaintiff's experts' specific causation theories and methodologies, the result is the same. The Court should exclude the Plaintiff's expert's specific causation opinions because Plaintiff's experts' methodologies are unreliable and fail to satisfy Rule 702.

A. There is a generally accepted and reliable method for determining whether exposure to a particular substance did in fact cause a plaintiff's injuries.

To prove toxicological cause and effect, one must prove that (1) there are not alternative causes of the harm, *see Sorensen*, 31 F.3d at 649, and (2) actual levels of exposure of a chemical

were present at doses that are known to cause the particular type of harm, *see Wright*, 91 F.3d at 1106 ("[A] plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover.").

Only if experts can demonstrate both of these factors can they reliably opine on a toxicological cause and effect (*i.e.*, that a particular chemical caused a particular injury). This is particularly true in a case involving allegations of pre-birth injury. Indeed, in a case involving allegations of pre-birth injuries from the mother's alleged exposure to Dursban, the Eighth Circuit *affirmed* the district court's exclusion of the plaintiffs' experts—not only on general causation, but also on specific causation:

> In determining the cause of birth defects it is necessary not only to rule in a particular possible cause but also to rule out other possible causes. The plaintiffs have failed to do either in this case. . . .
>
> To establish specific causation in the case it was incumbent upon plaintiffs to provide evidence from which a jury could responsibly assess the level of the exposure of Mrs. Smits to Dursban while she worked at the bank. How much of the chemical got into the ambient air where it might be inhaled? What amounts of the chemical settle on surfaces which might be touched by Mrs. Smits (dermal exposure)? How long would these effects last?
>
> The plaintiffs must also provide some evidentiary basis for the jury to assess the dose of the chemical taken in by Mrs. Smits and also the dose that reached the fetus.

Nat'l Bank of Commerce (Smits), 965 F. Supp. at 1520; *see also Sorensen*, 31 F.3d at 649 (affirming exclusion of plaintiffs' experts since they followed "no scientific principles" in opining that the mother's alleged exposure to chlorpyrifos caused the child's birth defects); *Hannan v. Pest Control Servs., Inc.*, 734 N.E.2d 674, 680-83 (Ind. Ct. App. 2000) (excluding plaintiff's experts' medical causation opinions in a Dursban exposure case because the experts

failed to evaluate alternative causes of the alleged adverse health effects and failed to analyze exposure and dose).

In this case, Plaintiff's experts' methodologies are inherently speculative and unreliable for their failure to "rule out" alternative causes and failure to consider exposure, dose, and dose/response.

B. Plaintiff's experts failed to "rule out" alternative causes for T.J.'s cerebral palsy and resulting neurodevelopmental delay; this failure renders their specific causation testimony inherently unreliable and inadmissible.

The first step of a reliable methodology in determining specific causation is that an expert must demonstrate that he has properly "ruled out" all alternative causes of a particular effect. Specifically, Plaintiff's experts' specific causation methodologies are unreliable because Plaintiff's experts' fail to rule out what T.J.'s treating physicians and other experts considered the primary cause of his cerebral palsy: prematurity and the large tumor, or chorioangioma, on T.J.'s umbilical cord.

1. A reliable methodology for determining specific causation requires that the expert rule out, in a systematic and reliable manner, alternative causes of the harm.

To determine specific causation, an expert must properly "rule out" all alternative causes of a particular effect.[9] *See, e.g.*, Rule 702 advisory committee notes (in determining reliability,

[9] This "ruling out" of alternative causes is also part of the "differential diagnosis" methodology used by some experts to determine a causal effect. *See Turner*, 229 F.3d at 1208. In addition to "ruling out" alternative causes, an expert must "rule in" the particular causal effect. *See id.*; *see also Cavallo v. Star Enter.*, 892 F. Supp. 756, 771 (E.D. Va. 1995) ("[T]he final, suspected 'cause' remaining after this process of elimination must *actually* be *capable* of causing the injury. That is, the expert must 'rule in' the suspected cause as well as 'rule out' other possible causes."). This "ruling in" requires an analysis of general causation and also the particular dose, exposure, and dose-response of the plaintiff. To "rule in" a particular cause, the substance must be capable of causing the particular effect, and the plaintiff must have been exposed to enough of a dose and exposure level of the substance to have caused harmful effects. *See, e.g.*, *Wright*, 91 F.3d at 1106.

courts should consider "[w]hether the expert has adequately accounted for obvious alternative explanations"); *Glastetter*, 252 F.3d at 989; *Turner*, 229 F.3d at 1208 (affirming exclusion of expert who did not "systematically rule out all other possible causes"); *Claar v. Burlington N. R.R. Co.*, 29 F.3d 499, 502 & n.3 (9th Cir. 1994) (even though expert stated that he ruled out other causes for Plaintiff's injuries, testimony about causation excluded because that "ruling out" lacked a basis in fact and was unreliable).

Although the "ruling out" of alternative causes need not be "carried to quixotic extreme," *see Lauzon*, 270 F.3d at 693, "ruling out" alternative causes itself requires a reliable methodology. *See, e.g.*, *Nelson v. Am. Home Prods. Corp.*, 92 F. Supp. 2d 954, 970 (W.D. Mo. 2000) ("To be admissible under *Daubert*, however, the differential diagnostic process of considering and eliminating other possible causes of a disease must still be based on sufficiently reliable methodology."). Indeed, Eighth Circuit law is crystal-clear that the "ruling out" must be reliable, proper and systematic. *See Turner*, 229 F.3d at 1208-09 (discussing a "reliable differential diagnosis," a "proper differential diagnosis," and excluding an expert because he did not "*systematically* rule out all other possible causes." (emphasis added)).[10]

An expert's mere statement that she has considered, and rejected, alternative causes is nothing more than an improper *ipse dixit.* Rather, the expert must demonstrate how and why each plausible alternative cause of injury was rejected. *See Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202 (4th Cir. 2001) ("[I]f an expert utterly fails to consider alternative causes or fails to offer an explanation for why the proffered alternative cause was not the sole cause, a district

[10] *Kudabeck v. Kroger Co.*, 338 F.3d 856 (8th Cir. 2003), does not hold to the contrary. Rather, in *Kudabeck*, there was no possible "cause" that the expert overlooked. *See id.* at 861. The Eighth Circuit's decision in *Kudabeck* confirms that the expert in that case did in fact perform a *reliable* differential diagnosis using a *reliable* methodology, contrary to what Drs. Bearer and Abou-Donia did in this case.

court is justified in excluding the expert's testimony."). Merely considering literature and rejecting it out of hand is also inadequate. *See id.* at 202-03 ("Simply asserting that he read two articles . . . and rejected them as unpersuasive is insufficient. Such a practice . . . renders his opinion . . . little more than speculation.").

In another case involving alleged chemical *in utero* exposure, the Eighth Circuit confirmed that *post hoc* rationalization to justify the end result is improper:

> Instead of reasoning from known facts to reach a conclusion, the [excluded] experts here reasoned from an end result in order to hypothesize what needed to be known but what was not. . . . [S]uch reasoning cannot apply here where several possible causes could have produced one effect.

Sorensen, 31 F.3d at 649; *see also Stibbs v. Mapco, Inc.*, 945 F. Supp. 1220, 1224 (S.D. Iowa 1996) (quoting *Sorensen* and excluding experts because their opinions were litigation-driven).

2. Dr. Bearer's method for "ruling in" and "ruling out" other causes is unreliable.

Dr. Bearer's method failed to consider plausible, alternative causes for T.J.'s cerebral palsy and resulting neurodevelopmental delay. Her failure to both "rule in" and "rule out" these other causes renders her testimony speculative and unreliable, and therefore excludable under Rule 702.

(a) Dr. Bearer's method ignored T.J.'s treating physicians, who, independent of litigation, noted that T.J.'s prematurity and the large tumor on his umbilical cord caused his cerebral palsy and resulting neurodevelopmental delay.

A review of T.J.'s medical records establishes the inherent unreliability of Plaintiff's methodology in establishing specific causation, because Plaintiff's experts ignore—and cannot rule out—what T.J.'s treating physicians have deemed to be the cause of his cerebral palsy and resulting neurodevelopmental delay. Specifically, T.J.'s treating physicians, independent of any

litigation, have noted that T.J.'s prematurity and the large chorioangioma on his umbilical cord caused his cerebral palsy and neurodevelopmental delay. Of note:

- Dr. Jill Meilahn recorded on November 29, 1994 that T.J.'s spastic diplegic and cerebral palsy were "secondary to prematurity." Ex. I to Scialli Decl.
- Dr. Emily Gavin wrote on May 20, 1997 that T.J. had a history of "cerebral palsy and spastic quadriplegia which was secondary to having suffered a chorioangioma of his umbilical cord in utero." Ex. J to Scialli Decl. She also wrote that the "prematurity and chorioangioma tumor of the umbilical cord result[ed] in cerebral palsy, spastic quadriplegia, and developmental delay." *Id.* at 3.
- Dr. David Moore recorded on October 24, 2006 that T.J. had "congenital spastic quadriparesis secondary to prematurity and umbilical tumor." Ex. K to Scialli Decl.

These opinions of T.J.'s treating physicians are supported by T.J.'s medical records, including objective evidence that he was deprived of oxygen *in utero*, and the medical literature, as explained below, which universally acknowledges prematurity as the most likely cause of cerebral palsy. In contrast, Dr. Bearer opines that there is no other cause for T.J.'s cerebral palsy and resulting neurodevelopmental delay other than his alleged exposure to chlorpyrifos. Supp. Bearer Rep. ("Differential diagnosis excludes, to a reasonable medical probability, other causes of [T.J.]'s condition."). This "ruling out" by Dr. Bearer lacks any reliable methodology, and her testimony must be excluded on these grounds.

(b) Dr. Bearer's methodology in "ruling out" T.J.'s prematurity as a primary cause of his cerebral palsy is unreliable and cannot withstand scrutiny.

Dr. Bearer conclusorily states that she has "ruled out" prematurity as the cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay. In both her initial expert disclosure and her supplemental report, she does not even *mention* T.J.'s prematurity. *See* Bearer Rep.; Supp. Bearer Rep. In her deposition, she offers no explanation for why she "ruled out" prematurity, other than her personal experience:

> Q: Do you agree that [T.J.]'s prematurity could be the cause of the cerebral palsy?
>
> A. I don't think so.
>
> Q: How do you rule out his prematurity as a cause of the cerebral palsy?
>
> A. In 32 week infants with very transient respiratory distress, we don't see cerebral palsy as being a sequela.[11] He has the same chance as a term baby for having CP.

Bearer Dep. at 136.

Dr. Bearer's method is scientifically invalid for several reasons. First, she admitted that her personal and professional experience does include not diagnosing children with cerebral palsy. *See* Bearer Dep. at 89. Her clinical experience involves seeing children in the neonatal intensive care unit, and does not typically include children older than twelve months of age. *Id.* at 67-68. Even if she saw infants with the signs and symptoms of cerebral palsy, she would refer those children to a pediatric neurologist. *Id.* at 89-90. This is not a situation where Dr. Bearer has conducted a typical and reliable medical differential diagnosis based on her "experience with hundreds of patients." *See Heller*, 167 F.3d at 155.[12] Rather, in her clinical practice, Dr. Bearer

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[11] A sequela is "an aftereffect of disease, condition or injury," or a "secondary result." Merriam-Webster Online, Sequela, at http://www.merriam-webster.com/dictionary/sequela.

[12] Even vast clinical experience of a treating physician (which Dr. Bearer does not have in this case) does not render a treating physician's methodology reliable. As explained by one district court:

> Doctors in their day-to-day practices stumble upon coincidental occurrences and random events and often follow human nature, which is to confuse association and causation. They are programmed by human nature and the rigors and necessities of their clinical practices to conclude that temporal association equals causation, or at least that it provides an adequate proxy in the chaotic and sometimes inconclusive world of medicine. This shortcut aids doctors in their clinical practices because their most important objective day-to-day is to help their patients and "first, do no harm," as their Hippocratic oath requires. . . . The Court also does not question that the methodology Dr. Kulig discussed at the *Daubert* hearing serves him well every day in the clinical practice of medicine. Dr. Kulig obviously is an exceptionally qualified practitioner, and the Court found him to be a very credible witness in this regard. Unfortunately, his clinical

sees *infants*. She does not see, treat or diagnose children with cerebral palsy, let alone children with cerebral palsy because of an environmental exposure, and further let alone children with cerebral palsy because of an alleged exposure to pesticides or, even more specifically, organophosphates. *See* Bearer Dep. at 102-103 (discussing how Dr. Bearer, in her twenty years of clinical practice, had seen "probably 10" cases of birth injuries involving a suspected chemical exposure cause, none of which involved chlorpyrifos or organophosphorate pesticides). Given this lack of experience with cerebral palsy, it is not surprising that Dr. Bearer does not "see" prematurity at 32 weeks as a cause for cerebral palsy because that is not her area of expertise.

Second, Dr. Bearer's own website confirms that the etiology of cerebral palsy in premature infants "remains poorly understood." Bearer website, http://neurowww.cwru.edu/faculty/bearer/index, Ex L to Eaton Decl. How can she exclude prematurity as a cause and include alleged chlorpyrifos exposure when she concedes outside of litigation that the etiology of cerebral palsy is poorly understood?

Third, in contrast to Dr. Bearer's blanket, unsupported assertion that "we don't see" it, there is substantial published medical literature linking the primary cause of cerebral palsy to prematurity. *See* Scialli Rep at 4; Graham Rep. at ¶ 30. For example, a recent epidemiological study of 2901 live births measured the incidence of cerebral palsy among children born very preterm, including children born at 32 weeks gestational age, as was T.J. The study concluded:

> Another important finding is that the cerebral palsy rate in children born at 31-32 weeks remained high in our study. Since these children accounted for more than half of very preterm survivors, a third of cases of cerebral palsy were recorded in children born at 31-32 weeks. Thus, the neurological outcome of infants born at or after 30 weeks should not be ignored.

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impression is not the sort of scientific methodology that *Daubert* demands.

Siharath v. Sandoz Pharms. Corp., 131 F. Supp. 2d 1347, 1372 (N.D. Ga. 2001).

Larroque et al, Neurodevelopmental Disabilities and Special Care of 5-Year-Old Children Born Before 33 Weeks of Gestation (the EPIPAGE study): a longitudinal cohort study, 317 Lancet 813, 819 (2008), Ex. L to Scialli Decl.

Likewise, a Swedish case-control study of 1746 cases of cerebral palsy found a substantial link between cerebral palsy and gestational age. The odds ratio for children born at the gestational age of 32-36 weeks was 3.9—meaning it is approximately four times as likely for a child born 4-8 weeks premature to develop cerebral palsy. *See* Thorngren-Jerneck & Andreas Herbst, Perinatal factors associated with cerebral palsy in children born in Sweden, 108 Obstetrics & Gynecology 1499, table 3 (2006), Ex. M to Scialli Decl. T.J. was born at 32 weeks gestation, the very *low end* of that group and thus the most likely to develop cerebral palsy.

Dr. Bearer's methodology included consciously ignoring published, peer-reviewed literature linking prematurity and cerebral palsy. Dr. Bearer's methodology of relying on her "experience" that she just doesn't "see it" is flawed and unreliable. She had no scientifically valid basis or explanation for ignoring prematurity as a cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay.

(c) Dr. Bearer's methodology in "ruling out" the large umbilical cord tumor as a primary cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay is unreliable.

T.J.'s physicians also attributed his cerebral palsy and resulting neurodevelopmental delay to the chorioangioma on T.J.'s umbilical cord.[13] Again, Plaintiff's experts have *not* rendered an expert opinion that exposure to chlorpyrifos is capable of causing or did in fact cause

[13] The chorioangioma could have caused T.J.'s premature birth. It is irrelevant whether T.J.'s cerebral palsy and resulting neurodevelopmental delay was caused by the chorioangioma, his premature birth, or some combination of the two. Neither chorioangioma nor premature birth is linked to chlorpyrifos exposure. *See* Scialli Rep. at 4; *see also supra* Part II (general causation discussion).

the chorioangioma. Rene Junk's physicians first diagnosed the chorioangioma at 23 weeks, and noted its significant size: 6.4 cm x 6.1 cm x 4.1 cm. The specialist wrote: "I have seen similar cord abnormalities before, but never on this scale. I cannot therefore hazard a guess as to the implications it has for the pregnancy." July 6, 1992 Letter from Dr. Carl Weiner, Ex. N to Scialli Decl.

For the next nine weeks, the umbilical cord tumor grew with the pregnancy. At 32 weeks, an ultrasound measured the tumor at 7.1 cm x 7.5 cm x 8.5 cm. Graham Rep. at ¶ 49. After delivery, the chorioangioma and umbilical cord weighed 400 grams, which meant that, at birth, T.J.'s placenta and umbilical cord tumor weighed almost half as much as T.J. did. *Id.* at ¶ 50.

A tumor the size of the chorioangioma in T.J.'s umbilical cord produces severe effects. Chorioangioma is associated with cardiac enlargement and stillbirth, and 35-37 % perinatal mortality. *See* Graham Rep. at ¶ 51; Scialli Rep. at 4. In addition, chorioangioma has been associated with cerebral palsy. *See* Harigaya et al, Premature infant with severe periventricular leukomalacia associated with a large placental chorioangioma: a case report, 22 J. Perinatology 252, 252 (2002), Ex. O to Scialli Decl. Chorioangiomas "that exceed 5 cm in diameter may be associated with maternal or fetal complications," and a 7 x 6 chorioangioma may be reported as a cause of cerebral palsy. *Id.* The chorioangioma on T.J.'s umbilical cord was even larger, measuring 7.1 cm x 7.5 cm x 8.5 cm. *See* Graham Rep. at ¶ 49.

Dr. Bearer dismisses the chorioangioma as causing T.J.'s cerebral palsy and resulting neurodevelopmental delay, but her methodology in reaching that conclusion is not peer reviewed or tested, and is contrary to the generally accepted belief in the scientific community. Dr. Bearer had never reviewed or seen a patient with a chorioangioma similar to the one in T.J.'s umbilical

cord. *See* Bearer Dep. at 94. She just simply concluded that "chorioangioma is only noted to cause hydrops fetalis and that is not related to neurodevelopmental delay or loss of brain tissue." *Id.* at 132. Plain and simple, Dr. Bearer is plainly incorrect in her assertion that chorioangioma is only noted to cause hydrops fetalis. *See* Scialli Rep. at 4, 10; Graham Rep. at ¶ 49-52; Ross Rep. at 4-6. Thus, Dr. Bearer's method is factually flawed and lacks a proper foundation.

The primary adverse effect of the chorioangioma in T.J.'s umbilical cord is that it reduced blood flow to T.J.'s brain, which decreased T.J.'s oxygen and caused his cerebral palsy and resulting neurodevelopmental delay. For example, one of T.J.'s treating physicians remarked: "The cord had chorioangioma which certainly compromised some of the blood supply during his intrauterine life." Apr. 24, 1995 Medical Record of Visit to Dr. Murali Srinivasan, Ex. P to Scialli Decl. The large mass on the umbilical cord reduced T.J.'s blood flow and narrowed the umbilical blood vessels. *See* Scialli Rep. at 4; Ross Rep. at 4-5 (noting the chorioangioma's effect of hypoperfusion, which is decreased blood flow through the brain). This reduced blood flow decreases the oxygen that is received by the fetal brain. *See* Graham Rep. at ¶ 51; Scialli Rep. at 4; Altshuler Rep. This lack of oxygen, prior to T.J.'s birth, caused his cerebral palsy and resulting neurodevelopmental delay. Dr. Ruth Nass, a pediatric neurologist, summarized this course in her expert report: "The birth history of the large chorioangioma may also have contributed to the development of cerebral palsy by causing decreased blood flow to the brain secondary to poor cerebral auto regulation in a preterm infant. Decreased blood flow to the brain can cause damage to the brain tissue putting the neonate at risk for cerebral palsy." Expert Report of Dr. Ruth Nass ("Nass Rep."), Ex. B to Declaration of Dr. Nass, App, Ex. 7. Dr. Altshuler also confirmed that "the features of reduced uteroplacental blood flow and elevated NRBCs [nucleated red blood cells] are significant risk factors for damage to the fetal brain,

postnatal cerebral palsy, and developmental delay.[14] Supp. Altshuler Rep.; *see also* Altshuler, *Placental Pathology and the Etiology of Fetal and Neonatal Brain Injury* in FETAL AND NEONATAL BRAIN INJURY (Stevenson et al., eds. 2003), Ex. N to Eaton Decl.

Medical doctors and experts use two different terms to describe this lack of oxygen, which was caused by the chorioangioma. T.J. suffered from *brain asphyxia in utero*, which means he had a condition of a severely deficient supply of oxygen to the brain prior to birth. As a result, after he was born, T.J. suffered from *hypoxia*, which means T.J. did not have enough oxygen getting to his organs.

(i) Dr. Bearer's method failed to account for evidence of brain asphyxia.

Evidence of the critical brain asphyxia and later hypoxia that T.J. suffered from is manifested in his elevated levels of nucleated red blood cells, or NRBCs. NRBCs are immature blood cells that are present when there is a deprivation of oxygen in the blood flow. The NRBCs attempt to "compensate" for a lack of oxygen. T.J.'s NRBC levels were 33, which are abnormally high. *See* Altshuler Supp. Rep., App.; Scialli Rep. at 3; Ross Rep. at 2; Graham Supp. Rep. at 2. Dr. Altshuler reviewed the tissue blocks from the placenta and explained the significance of the NRBCs and other findings:

> NRBCs signify that the fetus is attempting to compensate for hypoxia. [T.J.]'s NRBC count was 3.8 x 10^9 / L. That very high NRBC level is pathological. Elevated NRBCs most often result from reduced uteroplacental blood flow.
>
> Because the placenta showed ischemic changes it is within a reasonable degree of medical certainty that the fetus experienced clinically very significant hypoxia or asphyxia.

Altshuler Supp. Rep.

[14] Dr. Altshuler is a pediatric placental pathologist. He examined the blocks and tissues from Rene Junk's placenta to confirm there was evidence of brain asphyxia and other abnormalities. *See* Altshuler Rep.

Dr. Bearer's method did not include an assessment or even consideration of T.J.'s elevated NRBC levels. When confronted at the deposition, Dr. Bearer simply dismissed the elevated levels of NRBCs, opining without reasonable explanation that T.J.'s elevated NRBCs adequately compensated for the lack of oxygen T.J. received to his brain. *See* Bearer Dep. at 80, 143. This methodology of rejecting the high levels of NRBCs is unsound. T.J.'s NRBC levels are objective, concrete evidence that T.J. suffered from reduced uteroplacental blood flow, very significant hypoxia, and critical asphyxia. NRBCs may have been able to compensate for T.J.'s lack of oxygen, possibly preventing a fatal outcome that is present in many cases of brain asphyxia. However, NRBCs were simply unable to compensate fully T.J.'s chronic hypoxia *in utero.* Because the NRBCs are only immature blood cells, they could not prevent the permanent damage to T.J.'s sensitive premature brain cells. *See* Altshuler Supp. Rep.

In addition, Rene Junk's June 30, 1992 ultrasound (where the chorioangioma was discovered), showed an abnormally high systolic/diastolic (S/D) rate at 6.3 per pulsed Doppler when the fetus was a 23.9 weeks gestational age. Deposition of Anthony Scialli ("Scialli Dep."), Ex. O to Eaton Decl. This finding indicates an impairment of uteroplacental blood flow through the umbilical cord and/or through the placenta, suggesting that not enough blood is getting between the baby and the placenta. Scialli Dep. at 51-52. Dr. Bearer ignored this objective finding as well.

(ii) Dr. Bearer's method regarding evidence of hypoxia is scientifically invalid.

Following birth, T.J. suffered from hypoxia, or a critical lack of oxygen. Dr. Bearer simply dismisses the multiple signs of T.J.'s hypoxia in rushing to her causal opinion. In her report, she states, "[A]lso, no evidence of hypoxia at the time of delivery exists. His cord Ph was within normal limits, and his first blood gas at approximately 2 hours of life showed no acidosis

or base deficit, clearly eliminating hypoxia as a cause of his condition." Bearer Rep. Dr. Bearer admitted her confusion over the medical records of T.J.'s blood gas tests at birth, *see* Bearer Dep. at 41 ("Maybe I was confused . . . ."), and her methodology for ruling out hypoxia in the face of the following symptoms is unreliable:

Evidence of T.J.'s Hypoxia

- T.J.'s Apgar scores (a method used to summarize the health of newborn children) were below normal. He had a score of 5 at one minute, and a 6 at five minutes.

  See, e.g., Scialli Rep. at 3.

- T.J. required resuscitation efforts immediately after his delivery. He was incubated and required "bagging" of partial pressure oxygen.

  See, e.g., *id.*, at 3.

- T.J. had flaring of the nostrils, subcoastal retractings and grunting, and shallow breathing at birth. These are signs of respiratory distress. Dr. Bearer simply dismisses the effect of this respiratory distress, stating "I think I'm getting mixed up."

  See Bearer Dep. at 97, 139.

- T.J. had a blood gas test taken in his foot approximately fifteen (15) minutes after delivery. This foot stick pH was 7.19, which is below normal. The foot stick pH reflects T.J.'s critical lack of oxygen prior to birth.

  Dr. Bearer did not consider the foot stick blood gas test and relied instead on another blood gas test taken from T.J.'s umbilical cord two hours later when he was on supplemental oxygen. Dr. Bearer later admitted that T.J.'s foot stick pH was low, although she had not referenced this medical fact in her report or even understood when it was taken.

  See Ross Rep. at 2; Graham Rep. at ¶ 50; Supp. Altshuler Rep.; Bearer Dep. at 39-40, 85.

- T.J.'s umbilical cord not only contained the large tumor, but T.J.'s umbilical cord was also short, indicating lack of fetal growth.

  See Supp. Graham Rep. at 2.

T.J.'s evidence of hypoxia is especially relevant here because hypoxia during development of the brain can cause cerebral palsy and developmental delay. Supp. Scialli Rep. at 4.

Dr. Bearer's methodology in ruling out the chorioangioma, and subsequent hypoxia, as a cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay is simply that she does not have one. She rejects without a reliable explanation this cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay. Her methodology in concluding exposure to chlorpyrifos caused T.J.'s cerebral palsy and resulting neurodevelopmental delay is therefore unreliable.

(d) Dr. Bearer's litigation method is not consistent with the same intellectual rigor she utilizes in her practice.

Dr. Bearer's methodology for determining whether T.J.'s cerebral palsy and resulting neurodevelopmental delay were caused by his alleged exposure to chlorpyrifos lacks any sort of intellectual rigor, and does not rise to the same level of intellectual rigor that Dr. Bearer *herself* uses in her clinical practice. *Ahlberg v. Chrysler Corp.*, 481 F.3d 630, 635 (8th Cir. 2007); *see Kumho Tire*, 526 U.S. at 152 (holding that the objective of the gatekeeping requirement "is to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field"); Fed. R. Evid. 702 advisory committee notes (listing as a factor courts may consider "whether the expert is being as careful as he would be in his regular professional work outside his paid litigation consulting."). For example, a key piece of information in determining the cause of T.J.'s cerebral palsy and resulting neurodevelopmental delay is a pathological analysis of the placenta. Dr. Bearer admits that in her clinical practice, she works with a placental pathologist, Dr. Redline, "on a routine basis" to help determine the cause of an infant's medical problems. *See* Bearer Dep. at 78-79, 142. Yet in this case, she

initially did not even review the pathology reports. *See id.* at 77, 141-142. Only after DAS disclosed Dr. Altshuler, an expert in pediatric placental pathology, did Dr. Bearer even *review* the pathology report. Even then, she summarily concluded that the report "did not change [her] opinions" and that she did not consult with Dr. Redline or any other colleagues with expertise in placental pathology. *Id.* at 77, 142. Her methodology in forming an opinion with respect to causation thus differs dramatically from the methodology she follows in her typical clinical practice.

Dr. Bearer's methodology for "ruling out" alternative causes for T.J.'s cerebral palsy and resulting neurodevelopmental delay simply does not measure up to the standards of intellectual rigor she herself employs in her clinical practice.

3. Dr. Abou-Donia's opinions regarding "ruling out" alternative causes are inadmissible because he admits he is not qualified to render those opinions.

Dr. Abou-Donia's testimony "ruling out" other causes must be excluded, because he himself admits that he cannot opine as to other possible causes of T.J.'s cerebral palsy and resulting neurodevelopmental delay. Dr. Abou-Donia is not a medical doctor. He has a Ph.D. in agricultural chemistry and readily admits that he does not work with humans. Abou-Donia Dep. at 39, 59, 78. Dr. Abou-Donia admitted in his deposition that he was not "giving any opinions relating to medical causation":

Q. You have no other degrees, for example, in pharmacology, neurology, placental pathology or genetics, is that right?

A. No.

Q. Is that correct?

A. That's correct. I have a professor of pharmacology and neurobiology.

Q. And you're not a medical doctor, correct?

A. Correct.

Q. And you're not in this case, in the Junk case you're not giving any opinions relating to medical causation, is that correct?

A. That's correct.

Q. You realize your report says you're relying upon Dr. Bearer, the medical doctor the plaintiffs have disclosed?

A. Correct.

Id. at 38.

He further admitted he was not qualified to rule out alternative causes. *Id.* at 81 ("Q: But my question was, you agree you're not qualified as a Ph.D. agricultural chemist to rule out or exclude prematurity in [T.J.] as a cause of his cerebral palsy? A: That's true."); *see also id.* at 59 (discussing Dr. Abou-Donia's lack of research into cerebral palsy and chorioangioma); 86-87 (discussing Dr. Abou-Donia's lack of assessment as to whether the chorioangioma could have caused decreased oxygen levels to T.J. *in utero*); 91 (discussing Dr. Abou-Donia's lack of awareness of studies linking chlorpyrifos to chorioangioma).

Because Dr. Abou-Donia is not qualified to rule out T.J.'s prematurity and chorioangioma as the cause for T.J.'s cerebral palsy and resulting neurodevelopmental delay, he must not be allowed to testify that there are no plausible alternative causes for T.J.'s cerebral palsy and resulting neurodevelopmental delay.

C. Plaintiff's experts' methodologies are unreliable because they do not in any way quantify the level of exposure or dose of chlorpyrifos received and, ignore the dose-response relationship.

Even assuming that Dr. Bearer and Dr. Abou-Donia *could* testify that exposure to chlorpyrifos is capable of causing cerebral palsy and resulting neurodevelopmental delay, and further *could* testify that T.J.'s specific injuries were *not* caused by alternative causes, their testimony would be unreliable for the separate and independent reason that they impermissibly

use a flawed methodology for determining whether Rene Junk and/or T.J. was exposed to *enough* chlorpyrifos to cause T.J.'s cerebral palsy and resulting neurodevelopmental delay.

1. A reliable methodology considers exposure, dose and dose-response.

The second step in a reliable methodology for determining specific causation is a consideration of exposure, dose, and dose-response. Of course, the plaintiff must be exposed to the allegedly harmful substance before liability attaches. Equally true, however, is that the plaintiff must be exposed to *enough* of the allegedly harmful substance to cause injury. How much of a substance a person is exposed to is referred to as the "dose" of exposure.

A "central tenet[] of toxicology" is that " 'the dose makes the poison'; this implies that all chemical agents are intrinsically hazardous—whether they cause harm is only a question of dose. Even water, if consumed in large quantities, can be toxic." Federal Judicial Center, *The Reference Manual on Scientific Evidence* 403 (2d ed. 2000) ("Reference Manual") (footnote omitted). Put another way:

> Dose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect. Indeed, the basic dictum of toxicology was stated by the Sixteenth Century Physician/Philosopher, Paracelsus, considered the 'father of toxicology': *'All substances are poisonous—there is none which is not; the dose differentiates a poison from a remedy.'*

In re Accutane Prods. Liab., 511 F. Supp. 2d 1288, 1293 (M.D. Fla. 2007) (emphasis original) (quoting David Eaton, *Scientific Judgment and Toxic Torts-A Primer in Toxicology for Judges and Lawyers*, 12 J. L. & POL'Y 1, 11 (2003)).

The Eighth Circuit has adopted this fundamental, basic premise for use in determining the reliability of an expert's specific causation testimony in chemical exposure cases. In a chemical exposure case, a plaintiff must not only establish "the levels of exposure that are hazardous to human beings generally," but the plaintiff must also establish "the plaintiff's actual level of

exposure" to the allegedly harmful substance. *Wright*, 91 F.3d at 1106. The Eighth Circuit has elaborated:

> At a minimum, we think that there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered. We do not require a mathematically precise table equating levels of exposure with levels of harm, but there must be evidence from which a reasonable person could conclude that a defendant's emission has probably caused a particular plaintiff the kind of harm of which he or she complains before there can be a recovery.

Id. at 1107 (citation omitted). In *Wright*, although the plaintiffs could prove that they had been exposed to some amount of the allegedly harmful agent, they could not demonstrate that they had been exposed to *enough* of that agent to have caused harm. *Id.*

The Eighth Circuit rejects attempts by plaintiffs' experts to utilize methodologies that presume that the plaintiffs are exposed to *enough* of a dose simply because they suffered injury. "That inference turns scientific analysis on its head." *Sorensen*, 31 F.3d at 649; *see also Nat'l Bank of Commerce*, 191 F.3d at 864 (affirming exclusion of plaintiffs' expert witnesses because they had "no scientific knowledge or information as to the level of AFM exposure that would subject a person who breathes in an aerosolized milk containing AFM to an appreciable risk of laryngeal cancer"); *Mitchell v. GenCorp., Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) ("[A] plaintiff must demonstrate the levels of exposure that are hazardous to human beings generally . . . before he or she may recover." (internal quotation marks omitted)); *Allen v. Penn. Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) ("Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case.").

After evaluating exposure (including amount, duration, and frequency) and the resulting dose, a reliable methodology for determining specific causation identifies a dose-response relationship for the particular chemical by analyzing medical literature or other scientific sources

to determine whether a relationship exists between the alleged harm and the exposure levels and dose in question. *See Nat'l Bank of Commerce (Smits)*, 965 F. Supp. at 1524; Federal Judicial Center, Reference Manual at 406-09, 422-23. The correlation between dosage and the response it engenders in a living organism is called the "dose-response" relationship:

> The dose-response relationship is a relationship in which a change in amount, intensity, or duration of exposure to an agent is associated with a change—either an increase or decrease—in risk of disease. The expert who avoids or neglects this principle of toxic torts without justification casts suspicion on the reliability of his methodology.

McClain v. Metabolife Int'l, Inc., 401 F.3d 1233, 1241-42 (11th Cir. 2005) (citations, quotation marks and brackets omitted).[15] The Eighth Circuit has confirmed that the detail need not be mathematically precise, but there must be *enough* detail for plaintiffs to "make a threshold showing that he or she was exposed to toxic *levels* known to cause the *type* of injuries he or she suffered." *Mattis v. Carlon Elec. Prods.*, 295 F.3d 856, 860-61 (8th Cir. 2002) (emphases added).

Neither Dr. Bearer nor Dr. Abou-Donia followed the standard toxicological methodology, and their failure to do so renders their testimony inadmissible. Instead, the Plaintiff's experts appear to be working backwards—T.J. has cerebral palsy and resulting neurodevelopmental delay and was allegedly exposed to *some* amount of chlorpyrifos; therefore he must have been exposed to "enough" chlorpyrifos to cause his cerebral palsy and resulting neurodevelopmental delay. As a matter of sound science, and as a matter of law, this approach is

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[15] *See also Wills v. Amerada Hess Corp.*, 379 F.3d 32, 49 (2d Cir. 2004) (affirming the district court's rejection of single-exposure theory of causation in toxic tort cases as controversial and unreliable science, as opposed to the generally accepted dose-response relationship method); *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278-79 (5th Cir. 1998) (en banc); *Wintz v. Northrop Corp.*, 10 F.3d 508, 513 (7th Cir. 1997). In other words, "[i]f the level of exposure was below this no observable effect, or threshold, level [the level that is incapable of causing harm], a relationship between the exposure and disease cannot be established." Reference Manual at 426.

untenable. As such, expert opinions that chlorpyrifos caused T.J.'s cerebral palsy and resulting neurodevelopmental delay are unreliable and excludable under Fed. R. Evid. 702.

2. Dr. Bearer's assumptions regarding exposure, dose, and dose-response render her specific causation methodology unreliable.

Dr. Bearer explains that she just *assumed* that T.J.'s alleged exposure was "significant." Bearer Dep. at 60, 122. ("I assumed it was a high number from what I heard."). Indeed, Dr. Bearer's second report's discussion of exposure and dose merely quotes and parrots Dr. Abou-Donia's report, *see* Supp. Bearer Rep., and Dr. Bearer admitted in her deposition that she simply relied on Dr. Abou-Donia's report, *see* Bearer Dep. at 61-62, 156, 163-164. Dr. Bearer has *no* knowledge of how much chlorpyrifos was applied at the Junk household, what type of application was used, or even how T.J. allegedly received a dose, in any amount, of chlorpyrifos. For example, she engaged in the following colloquy during her deposition:

Q: In forming your opinions in October of 2005 did you calculate or estimate the exposure or dose levels of Rene to chlorpyrifos?

A: I did not, no.

Q. Prior to October of 2005 did you estimate or calculate the exposure or dose levels of chloropyrifos to [T.J.] in utero?

A: In utero, no, I did not. Actually can you repeat the question, it was estimate, right?

Q: Estimate or calculate?

A: I didn't calculate. From what I heard, I assumed, which I guess is not estimate, estimate means an exact number, right, I didn't estimate. I assumed it was a high exposure from what I heard.

Q: You testified previously you assumed it was significant?

A: Yes, I'm sticking with that.

Q: Did you have any understanding of the type or method of application used by Terminix inside the home?

A: I did not.

Q: Were you aware of the quantity of product actually used inside the home prior to [T.J.]'s birth?

A: I was not.

Q: Did you review the Dursban product labels at any point in time prior to preparing your October 28, 2005 letter?

A: I did not. Can I take a break?

Bearer Dep. at 120-121. Likewise, she admitted she made no assessment of the duration of alleged exposure to T.J., *id.* at 167; she admitted she made no assessment of whether or not the exposure levels declined over time, *id.* at 168; and she had no knowledge of the dermal absorption rate of chlorpyrifos, *id.* at 171-172. This lack of knowledge renders her methodology for determining specific causation unreliable.

Dr. Bearer simply ignores dose, exposure levels, and the dose-response relationship. She is unfamiliar with the studies she cites, and cannot quantify the levels used within them. This lack of appreciation for exposure and dose is one of the many reasons why an Ohio court refused to admit Dr. Bearer's testimony in a chemical exposure case. *See Kerns vs. Hobart Bros. Co.*, Case No. 05-235 (Miami County, Ohio Oct. 3, 2007), Order Granting Defendants' Motion to Exclude the Testimony of Dr. Holland and Dr. Cynthia Bearer at 4 (excluding Dr. Bearer because, *inter alia*, she "failed to consider dose or exposure levels in forming [her] opinions"), Ex. M to Eaton Decl.

3. Dr. Abou-Donia's methodology regarding exposure, dose, and dose-response is unreliable.

Dr. Abou-Donia fails to offer much more detail than Dr. Bearer as to the exposure levels and absorbed dose. Dr. Abou-Donia was able, at one point, to state that Dursban L.O., containing 0.25 % chlorpyrifos, was applied at the Junk home. *See* Abou-Donia Rep. at 2; Abou-Donia Dep. at 89. Yet his report also contains a substantial section on Dursban TC, an

entirely different formulation with a different application method and concentration, which was never applied at the Junk home.[16] Abou-Donia Rep. at 7.

Like Dr. Bearer, Dr. Abou-Donia admits he made no specific findings as to levels of exposure or the dose absorbed by T.J. He made no specific assessment of the number of days Rene Junk was home during her pregnancy with T.J. when there were applications. Abou-Donia Dep. at 102-103. Likewise, he did not calculate the inhalation, dermal, or ingestion levels of chlorpyrifos during Rene Junk's pregnancy, and he did not calculate the dose that Rene Junk or T.J. received, whether through swallowing (ingestion), breathing (inhalation), or absorption through the skin (dermal). *See id.* at 103-104.

Dr. Abou-Donia simply assumed that the ambient exposure levels, and the absorbed dose, were sufficient to cause injury. He makes this assumption *even while* recognizing that there *are* standard reactions to exposure to massive quantities of chlorpyrifos that neither Rene Junk nor T.J. suffered from:

> Q: [Y]ou would expect to see signs and symptoms of an acute cholinergic crisis like salivation or lacrimation?
>
> A: That depends on the dose. If she were exposed to a higher dose, you would have seen that. But what she was exposed to was enough dose that actually caused her toxicity.

Id. at 128. Dr. Abou-Donia admits that Rene Junk was not exposed to *enough* chlorpyrifos to manifest the symptoms of an overexposure to chlorpyrifos. His "methodology" in concluding that Rene Junk was exposed to "enough dose" to cause T.J.'s cerebral palsy and resulting neurodevelopmental delay is simply that she must have been. This is unreliable and should be

[16] The application at the Junk home was a crack and crevice treatment of Dursban L.O. where the applied concentration contained only 0.25 % chlorpyrifos. In contrast, Dursban TC is a more concentrated termiticide that licensed technicians apply subterraneanously under the slabs of new home construction in much larger quantities. *See, e.g.*, Dursban TC label, available at http://oaspub.epa.gov/pestlabl/Ppls. getimage?imgid=162459.

excluded. *Sorensen*, 31 F.3d at 649 ("Instead of reasoning from known facts to reach a conclusion, the [excluded] experts here reasoned from an end result in order to hypothesize what needed to be known but what was not."); *Stibbs v. Mapco, Inc.*, 945 F. Supp. 1220, 1224 (S.D. Iowa 1996) (quoting *Sorensen* and excluding experts because their opinions were litigation-driven); *O'Conner v. Commonwealth Edison Co.*, 807 F. Supp. 1376, 1396 (C.D. Ill. 1992) ("[Plaintiff's expert] presumed that the cataracts were radiation induced, and then presumed that the plaintiff must have somehow been exposed to a high enough dose to exceed the threshold in order to have caused the cataracts, thereby justifying his initial diagnosis. This is circular reasoning."), *aff'd*, 13 F.3d 1090 (7th Cir. 1994).

(a) Dr. Abou-Donia, with a Ph.D. in agricultural chemistry, cannot reliably opine that Rene Junk's and T.J.'s alleged "symptoms" are "consistent with" exposure to chlorpyrifos.

Without any estimation of exposure or dose, Dr. Abou-Donia, who is not a medical doctor and whose area of expertise is agricultural chemistry, purports to offer an opinion that T.J. must have been exposed to enough chlorpyrifos to cause his cerebral palsy and resulting neurodevelopmental delay because, in essence, his mother was nauseous during pregnancy and he was "fussy" as an infant. These symptoms, according to Dr. Abou-Donia, are sufficient to allow him to "reliably" opine that T.J. was exposed to enough chlorpyrifos to cause cerebral palsy and resulting neurodevelopmental delay.

Dr. Abou-Donia is not qualified to testify regarding medical symptoms and whether those symptoms were indicative of chemical exposure or caused by something entirely separate from the alleged exposure. *See Housley v. Orteck Int'l, Inc.*, 488 F. Supp. 2d 819, 827 (S.D. Iowa 2007) (excluding expert who was not qualified on the particular subject matter of his "expertise"). Indeed, Dr. Abou-Donia admits nearly as much:

Q: And you're not in this case, in the Junk case you're not giving any opinions relating to medical causation, is that correct?

A: That's correct.

Abou-Donia Dep. at 38. Dr. Abou-Donia seems to draw a distinction between medical causation and whether a symptom is indicative of exposure or reflective of another cause, but that distinction is one without a difference. *See id.* at 46-47. A toxicologist without clinical experience examining patients is not qualified to rule out pregnancy, as opposed to exposure to chlorpyrifos, as the cause of Rene Junk's nausea throughout her pregnancy. *See, e.g.*, *Marmo v. Tyson Fresh Meats*, 457 F.3d 748, 758 (8th Cir. 2006) (affirming exclusion of toxicologist who did not examine plaintiff nor inquire about the plaintiff's other potential chemical exposures; noting that toxicologists are not necessarily *qualified* to render an opinion as to medical symptoms).

(b) Dr. Abou-Donia's method for determining Rene Junk and T.J.'s "symptoms" are consistent with the acute over exposure is unreliable and lacks any factual basis.

Even assuming a Ph.D. in agricultural chemistry may testify regarding medical symptoms, the fallacies in Dr. Abou-Donia's methodology regarding exposure and dose levels should be readily apparent. Dr. Abou-Donia is reasoning backward, starting from the hypothesis that T.J. *must* have been exposed to enough chlorpyrifos to cause his cerebral palsy and resulting neurodevelopmental delay. Moreover, the "symptoms" to which he points collapse like a house of cards when examined more closely.

Dr. Abou-Donia first attempts to explain the exposure T.J. "must have had" *in utero* by pointing to his mother's "symptoms" during pregnancy. The *only* symptoms to which Dr. Abou-Donia points to demonstrate exposure to chlorpyrifos are:

Dr. Abou-Donia's Purported "Evidence" of Rene Junk's and T.J.'s Exposure	**Correct Factual Foundation**
(1) Rene Junk was exposed after the first application Abou-Donia Rep. at 3.	(1) Rene Junk admitted that she spent three to four long weekends visiting her parents in La Crescent, Wisconsin, and it was possible she was away from the Junk home during the initial application. Rene Junk Dep. at 50, 183. Deposition of Dean Junk ("Dean Junk Dep.") at 43-44, Ex. P to Eaton Decl.
(2) Rene Junk "woke up the night after spraying and vomited, consistent with Dursban-induced cholinergic neurotoxicity." Abou-Donia Rep. at 3 (emphasis deleted).	(2) Rene Junk explained that she did *not* vomit until the seventh month of pregnancy, approximately four or five months *after* Dr. Abou-Donia attributed Rene Junk's "vomiting" to exposure to Dursban. Rene Junk Dep. at 40. Dr. Abou-Donia did not review the Junks' depositions to confirm his testimony. Abou-Donia Dep. at 99.
(3) Rene Junk "was in the first trimester and experienced some nausea, and some spotting." Abou-Donia Rep. at 3.	(3) Rene Junk testified that she experienced "spotting" around March 6, 1992, *before* the first application of chlorpyrifos on March 27. Rene Junk Dep. at 84-85; Ex. 10 to Rene Junk Dep, Ex. Q to Eaton Decl. "Nausea," "spotting" and "vomiting" during pregnancy are normal symptoms that pregnant women often experience; Dr. Abou-Donia offers no explanation as to why these "symptoms" are not just normal manifestations of pregnancy. *See, e.g.*, Supp. Scialli Rep. at 22-23; *see also* Abou-Donia Dep. at 47, 49.

(4) Application on July 9, 1992: On August 11, 1992, more than a month after this application, Dr. Abou-Donia reports that Rene Junk has diarrhea. "Diarrhea is consistent with exposure to Dursban that was sprayed on July 9, 1992." Abou-Donia Rep. at 3 (emphasis deleted).	(4) Rene Junk testified that she experienced "spotting" around March 6, 1992, *before* the first application of chlorpyrifos on March 27. Rene Junk Dep. at 84-85; Ex. 10 to Rene Junk Dep, Ex. Q to Eaton Decl. "Nausea," "spotting" and "vomiting" during pregnancy are normal symptoms that pregnant women often experience; Dr. Abou-Donia offers no explanation as to why these "symptoms" are not just normal manifestations of pregnancy. *See, e.g.*, Supp. Scialli Rep. at 22-23; *see also* Abou-Donia Dep. at 47, 49.
(5) Application on July 9, 1992: T.J.'s heart was "slightly enlarged" on the date of delivery, August 25, 1992, and "remained increased" on August 29, 1992. Dr. Abou-Donia then opines (without explanation or citation) that "*in utero* Dursban exposure causes tachycardia with subsequent heart enlargement." Abou-Donia Rep. at 3.	(5) The enlarged fetal heart was likely due to congestive heart failure secondary to the chorioangioma, as diagnosed by T.J.'s physicians. Scialli Supp. Rep. at 23. Dr. Abou-Donia admits his opinion as to the enlarged heart is nothing more than speculation. Abou-Donia Dep. at 131-132.
(6) T.J. "was fussy with feeding" and "hard to hold." T.J. "is unusually fussy often, including concurrent with feedings." T.J. had "poor appetite and slight cough." T.J. had "[c]lear runny nose and red throat" Abou-Donia Rep. at 3-4.	(6) Dr. Abou-Donia admitted T.J.'s "symptoms" represent childhood illness and the normal behavior of an infant. Abou-Donia Dep. at 133-140. Dr. Abou-Donia admitted that he could not exclude other causes of an infant being "fussy" separate from alleged exposure to chlorpyrifos (*id.* at 138); admitted that the reports of "fussiness" often came a *month* after an application of Dursban L.O. in the Junk home (*id.* at 135); and admitted that he did not know if T.J. was "fussier or more fussy the day after an application as opposed to the day before or two days before" (*id.* at 134).

Dr. Abou-Donia's reliance on Rene Junk's "symptoms" comprises the totality of his evidence that T.J. was exposed to "enough" chlorpyrifos[17] *in utero* to cause his cerebral palsy and resulting neurodevelopmental delay. There is simply no factual foundation from which Dr. Abou-Donia can reliably opine that these "symptoms" prove exposure and causation, because many of the "symptoms" are non-existent or disproven by the record. Dr. Abou-Donia's methodology of proving "enough" exposure through random, non-specific symptoms consistent with pregnancy fails to pass muster under *Daubert* and the advisory committee note factors: it is not tested (and accordingly has no error rate); it is not subject to peer review; it is not generally accepted within the scientific community; it is developed solely for litigation; it unjustifiably extrapolates; it disregards alternative causes; it does not involve the same intellectual rigor as clinical practice; and it involves a discipline outside the expertise of Dr. Abou-Donia. *See* Fed. R. Evid. 702 advisory committee notes.

In this case, Dr. Bearer concedes that she has little familiarity with the subject of her proffered opinion. She further admits to relying on the report of Dr. Abou-Donia in formulating her opinion as to the effect of exposure to chlorpyrifos. Bearer Dep. at 155-56.[18]

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[17] Dr. Abou-Donia also mentions that "[s]ome of the solvents used in pesticide formulation can produce tumors" or "cause skin rash." Any opinion about the "solvents" used in "pesticide formulation" must be disregarded, however, as Dr. Abou-Donia had *no* knowledge of the particular solvents that were used in the Dursban L.O. formulation, nor did he assert any opinion about those solvents. *See* Abou-Donia Dep. at 88 ("Q: You're not rendering any opinions in this case about whether or not the solvents in the Dursban formulation caused any of [T.J.'s] problems? A: Since I don't know the solvents, I cannot tell."); *see also* Bearer Dep. at 169 ("Q: You did not review the labels, the warning *labels* for the Dursban products involved or the material safety data sheets in forming your opinions? A: I did not. Q: Are you aware of the specific Dursban formulation used in the home? A: I am not.").

[18] Another indication that Plaintiff's experts have developed their opinions solely for litigation is the fact that Plaintiff's experts impermissibly rely on each other's opinions. Although an expert witness may rely upon what another witness observes or reports, the expert may not rely upon what the other concludes. *See Bryte v. Am. Household, Inc.* , 429 F.3d 469, 477 (4th Cir. 2005) ("*Daubert* aims to prevent expert speculation, and our review of the record convinces us that [the

Plaintiff's attempt to establish causation through the novel science of Drs. Bearer and Abou-Donia is litigation-driven and based on little more than pure conjecture, and, accordingly, is inadmissible under Rule 702 and *Daubert*.

Rather than rely on calculations or estimations of exposure, Dr. Abou-Donia simply states that T.J. must have been exposed to enough chlorpyrifos to cause his cerebral palsy and resulting neurodevelopmental delay because he was "fussy" and had a runny nose. The methodology behind equating common symptoms of nausea for pregnant women and cold-like symptoms or fussiness for infants is fundamentally unreliable and presents the classically excludable *post ergo hoc propter hoc* reasoning. As explained by Dr. Borgert:

> [Plaintiff's experts] make such conclusions without any objective means of quantifying either the chlorpyrifos dose necessary to produce the claimed effects in humans or verification that [T.J.], in fact, received such a dose. Dr. Abou-Donia, for example, invokes circular reasoning, devoid of measurements, claiming that symptomology proves the alleged exposure just as the alleged exposure proves the cause of those symptoms. . . . Both plaintiff experts substitute their own imaginative theories of how chlorpyrifos could cause effects similar to the specific health conditions suffered by [T.J.], and how he could have received a sufficient dose of chlorpyrifos to cause harm, for verifiable, reproducible data demonstrating causes and requisite doses for his specific conditions in either humans or animals. These general methodological flaws are repeated time and again . . . .

Supplemental Expert Report of Dr. Christopher Borgert ("Supp. Borgert Rep.") at 4, Ex C to Declaration of Dr. Borgert, App., Ex. 5; *see also Sorensen*, 31 F.3d at 649; *Stibbs*, 945 F. Supp. at 1224; *O'Conner*, 807 F. Supp. at 1396, *aff'd*, 13 F.3d 1090 (7th Cir. 1994).

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plaintiffs' expert's] failure to independently evaluate [a possible alternative cause] cannot be reconciled with the reliability mandate."); *see also Ninth Ave. Remedial Group v. Allis-Chalmers Corp.*, 141 F.Supp. 957, 959 (N.D. Ind. 2001) ("[A]n expert may not testify as to opinions that are based on others' work in an effort to shield a vulnerable expert from cross examination.").

(c) Dr. Abou-Donia's research does not fit.

Likewise, the majority of chlorpyrifos research conducted by Dr. Abou-Donia does not "fit" with this case because of his failure to consider exposure and dose. Dr. Abou-Donia opines, without citation, that exposure to chlorpyrifos can cause "delayed neurotoxicity." To reach his conclusions regarding "delayed neurotoxicity," Dr. Abou-Donia has conducted tests on chickens. *See, e.g.*, Joint Neurotoxic Action of Chlorpyrifos and Safrotin – Final Report (undated), Ex. R to Eaton Decl. In these tests, Dr. Abou-Donia treated hens with 100 mg/kg of chlorpyrifos by oral gavage (a process of feeding liquids through a tube which passes into the stomach through the nose). Dr. Abou-Donia then supported life in the hens through injections of heroic doses of antidotes. The levels of the massive doses provided to the chickens, and the heroic life supporting measures required to keep the chickens alive, invalidates the applicability and relevancy of Dr. Abou-Donia's work to humans. There is no remote similarity between any alleged exposure to a human through crack-and-crevice applications and the artificial circumstances of exposure created by Dr. Abou-Donia.

It is this lack of "fit" between massive doses of chemicals in Dr. Abou-Donia's hen studies that led the District Court for South Carolina to exclude Dr. Abou-Donia's testimony. *See Geowey*, 866 F. Supp. at 1280-81. "Dr. Abou-Donia's conclusions are extrapolated from his work on chickens [and] cannot meet the threshold test of reliability under *Daubert* and therefore, may not be considered probative of causation." *Goewey v. U.S.*, 886 F. Supp. 1268, 1280-81 (D.S.C. 1995), *aff'd*, 106 F.3d 390 (4th Cir. 1997) (table, text at 1997 WL 35348, at *2 ("The district court first excluded the testimony of . . . Mohamed Abou-Donia, M.D. [*sic*]. We have carefully reviewed the record, and we agree the district court properly excluded this testimony.")), *cert. denied sub nom. Goewey v. Fluor Daniel, Inc.*, 522 U.S. 1045 (1998).

D. Plaintiff's experts' specific causation methods are litigation-driven and unreliable.

Plaintiff's experts' specific causation methodology is not borne out of any research or work that they do in their clinical or academic endeavors. Rather, their opinions as to whether exposure to chlorpyrifos in fact caused T.J.'s cerebral palsy and resulting neurodevelopmental delay are necessarily driven by this litigation. This is a "very significant fact to be considered." *Daubert II*, 43 F.3d at 1317; *see also* Fed. R. Evid. 702 advisory committee notes (courts may consider "[w]hether experts are proposing to testify about matters growing naturally and directly out of research they have conducted independent of the litigation, or whether they have developed their opinions expressly for purposes of testifying" (internal quotation marks omitted)).

Plaintiff's experts' analyses in this case demonstrate how their specific causation opinions do *not* flow from any independent research, but rather were developed solely for the purposes of litigation. For example, Dr. Abou-Donia's report includes a substantial discussion of Dursban TC. *See* Abou-Donia Rep. at 7. At his deposition, Dr. Abou-Donia initially did not back away from his understanding that Dursban TC was applied at the Junk home, despite the fact that there is absolutely *no* evidence Dursban TC has *any* role in this case whatsoever. *See* Abou-Donia Dep. at 90. ("Q: So in forming your opinions in this case back on June 12th of 2007, it was your understanding that Dursban TC was applied inside the Junk home? A. Whatever I received from them. Q: Well, your report says Dursban TC. A. Then that must be it."). During a break, however, Plaintiff's counsel informed Dr. Abou-Donia that only Dursban L.O. was utilized in the Junk home. *See id.* at 95 ("Q: So Mr. Bye at the lunch break confirmed with you that Dursban TC was not used in the Junk home. A. Correct."). Dr. Abou-Donia's

report, however, nonetheless still continues to discuss Dursban TC, and Plaintiff has not provided a supplemental report to correct this error.

Another bizarre error left uncorrected is a heading in Dr. Abou-Donia's report entitled "Correlation Between Exposure to **esfenvalerate** and **Jason's** condition." Abou-Donia Rep. at 9. The obvious inference is that Dr. Abou-Donia simply copied a report from another one of his many litigated cases and forgot to change the names of the allegedly harmful substance and the allegedly injured party.

Dr. Bearer's specific causation opinions have also been driven by litigation. For example, Plaintiff's counsel arranged for members of the Junk family, including T.J., to fly to Cleveland and meet with Dr. Bearer at the airport. This hour-long get-together took place on December 29, 2007, long after Dr. Bearer had formulated her opinions in the case. Nonetheless, Plaintiff's counsel asked Dr. Bearer to meet with the Junks, she assumed, so that she "could tell the jury that [she] had evaluated [T.J.] prior to trial." Bearer Dep. at 52. In her clinical practice, Dr. Bearer examines newborns. *Id.* at 52. She does not treat or see 14-year-olds, and her clinical practice certainly does not include one-hour meetings at an airport that do not include detailed clinical histories. *Id.* at 52, 57.

Because Plaintiff's experts' specific causation opinions are driven solely from litigation, it is necessary that their findings be independently validated. Generally speaking, if testimony was developed in consideration of litigation, it is less likely that the scientific methodology was subject to peer review or publication. *See, e.g.*, *Haggerty v. Upjohn Co.*, 950 F. Supp. 1160, 1164 (S.D. Fla. 1996) (excluding the expert's testimony because she had not conducted independent research, which rendered her testimony speculative). Therefore, it is less likely that the testimony is based upon reliable scientific methodologies. For this reason, if the expert's

testimony is not based upon research conducted independent of the litigation, the proponent of the expert testimony must corroborate the litigation-driven hypothesis—an expert's "bald assurance" that his or her methodology is scientific is unreliable and impermissible. *Daubert II*, 43 F.3d at 1316. Rather:

> If the proffered expert testimony is not based on independent research, the party proffering it must come forward with other objective, verifiable evidence that the testimony is based on 'scientifically valid principles.' . . . the proponent of expert scientific testimony may attempt to satisfy its burden through the testimony of its own experts. For such a showing to be sufficient, *the experts must explain precisely how they went about reaching their conclusions and point to some objective source -- a learned treatise, the policy statement of a professional association, a published article in a reputable scientific journal or the like -- to show that they have followed the scientific method, as it is practiced by (at least) a recognized majority of scientists in their field.*

Id. at 1318-19 (emphasis added).

Plaintiff's experts have no such independent validation of their specific causation methods. The methods used by Plaintiff's experts are entirely speculative and largely borne out of Plaintiff's experts' own imaginative theories, with no corroborating or validation information. Plaintiff's experts' testimony as to specific causation should be excluded under Rule 702.

IV. Because Plaintiff Has No Admissible Expert Testimony as to General or Specific Causation, DAS Is Entitled To Summary Judgment.

DAS is entitled to summary judgment because Plaintiff has no admissible expert causation testimony, for the reasons discussed above. Medical causation is an essential element of Plaintiff's claims against DAS that must be proven through admissible expert testimony. *See, e.g.*, *Benedict v. Zimmer*, 405 F. Supp. 2d 1026, 1033-34 (S.D. Iowa. 2006) ("Regardless of what sort of defect the [plaintiffs] allege, the element of causation, which is required in both claims, requires the presentation of expert evidence." (citation omitted)); *Doe ex rel Doe v. Baxter Healthcare Corp.*, 178 F. Supp. 2d 1003, 1017 (S.D. Iowa 2001) (holding that "whether a particular blood component was the likely cause of [the plaintiff's] HIV infection" required

expert testimony); *see also In re Baycol Prods. Litig.*, 321 F. Supp. 2d 1119, 1125 (D. Minn. 2004) ("[T]his Court joins with those courts that have held personal injury cases involving pharmaceuticals, toxins or medical devices involve complex questions of medical causation beyond the understanding of a lay person."); *Giddings v. Bristol-Meyers Squibb Co.*, 192 F. Supp. 2d 421, 423 (D. Md. 2002) (" Expert testimony is usually necessary since evidence relating to causation involves technical medical questions beyond common knowledge of laypersons, and . . . raises technical questions requiring expert testimony.").

Plaintiff's proffered medical causation expert opinion testimony is inadmissible and, therefore, insufficient to overcome summary judgment in favor of the Dow Defendants. *See, e.g.*, *Housley v. Orteck Int'l, Inc.*, 488 F. Supp. 2d 819, 834 (S.D. Iowa 2007) ("The failure to retain an appropriate expert witness or advance any evidence of a defect other than unsupported conclusions means Housley has not demonstrated a genuine issue of material fact on his product liability claims, in negligence, warranty, or strict liability."); *Nat'l Bank of Commerce (Smits)*, 956 F. Supp. at 1530-31 (as a "direct consequence" of the exclusion of plaintiff's experts, "summary judgment must likewise be granted" to the Dow Defendants), *aff'd*, 133 F.3d 1332 (8th Cir. 1998); *Cavallo v. Star Enter.*, 100 F.3d 1150, 1159 (4th Cir. 1996) (where court concluded that the bases of the doctors' opinions were not sufficiently established to warrant their admission into evidence, the court excluded their testimony and granted summary judgment).

Accordingly, upon the exclusion of Plaintiff's causation expert opinions from evidence, the Court should grant summary judgment in favor of DAS due to Plaintiff's lack of the required admissible expert testimony on the essential element of medical causation.

CONCLUSION

For the foregoing reasons, DAS' motion to exclude the expert testimony of Dr. Bearer and Dr. Abou-Donia should be granted, and summary judgment in favor of DAS should be granted, together with all other relief just and proper in the premises.

Respectfully submitted,

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CERTIFICATE OF SERVICE

The undersigned hereby certifies that a copy of the foregoing document has been served this 12th day of May, 2008, was filed electronically with the Clerk of Court to be served by operation of the Court's electronic filing system to the following counsel of record:

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